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EXTINCTION TESTS IN SCARLET FEVER*

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In 1918, Schultz and Charlton [1] first described the discutient phenomenon, which they obtained after the injection of 1 c.c. of normal or convalescent scarlet fever serum, intradermally, in a scarlet fever patient with a fully developed eruption. According to these writers, a blanching of the skin, together with a complete disappearance of the rash around the injection for an area of from 1 to 2 inches (2.5 to 5 cm.) in diameter occurred within from five to six hours after the injection, and persisted while the scarlatinal rash lasted. In their first series of fifty patients, forty-four gave positive reactions, with five doubtful, and one negative; while patients with other skin rashes when injected with the same serums gave uniformly negative reactions. These authors also pointed out that the injection of serum from patients in the exanthematous stage of scarlet fever did not produce this reaction. This phenomenon was investigated by Hainiss,[2] who in a series of nineteen cases, reports eleven positive and eight negative reactions.

Paschen [3] tested seventy-one cases, sixty-two of which were positive and nine negative, and explains the negative results by saying that the serum was used too late or not injected intracutaneously. He also found that the reaction did not occur with diphtheria antitoxin or with normal horse serum. Tron [4] compared fifty children in different stages of scarlet fever with ninety-nine controls, tested according to the Schultz-Charlton technic, and found an occasionally positive reaction, obtaining only ten definite reactions out of forty-seven frank scarlet fever cases. Steinkopf [5] obtained positive results in 83.7 per cent. of

* Received for publication, Nov. 26, 1923.

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1. Schultz-Charlton: Blanching of the Skin by Serum Injection in Scarlet Fever, Ztschr. f. Kinderh. **17**:328, 1918.

2. Hainiss, E.: Differential Diagnosis in Scarlet Fever, Monatsch. f. Kinderh. **15**:523, 1919.

3. Paschen, E.: Scarlet Fever Diagnosis, Dermat. Wchnschr. **68**:342, 1919.

4. Tron, G.: Induced Local Blanching in the Diagnosis of Scarlet Fever, Riforma med. **37**:55 (Jan. 15) 1921; abstr. J. A. M. A. **76**:899 (March 26) 1921.

5. Steinkopf, C.: Diagnostic Skin Phenomena in Scarlet Fever, Ztschr. f. Kinderh. **31**:132, 1921; abst. J. A. M. A. **78**:156 (Jan. 14) 1922.

forty-nine cases of scarlet fever, and states that the eruption does not merely blanch but that its elements retrogress. Hazelhorst [6] employed the test in fifty undoubted cases of scarlet fever and in thirteen cases of other skin diseases and found that the latter were consistently negative. He also noted that the reaction was negative in as many as 20 per cent. of the cases in which the diagnosis of scarlet fever was certain. He states that the test usually fails to be of any help in the doubtful cases of scarlet fever with slight fever, atypical throats and ill marked or transient rashes.

Mulsow [7] injected thirty patients with convalescent serum obtained from a scarlet fever patient from twenty-one to twenty-eight days after the rash appeared, and found that 53 per cent. gave positive and 10 per cent. questionable reactions. In another series of twenty cases, he injected normal serum and obtained 40 per cent. positive and 15 per cent. questionably positive reactions. He then injected thirty patients with serum taken during the acute stage of scarlet fever and obtained negative results except in one instance, in which the reaction was questionable. He noted the fact that heating the serum had no effect on its reaction. Rojo [8] states that in eighteen of his cases 78 per cent. of the reactions were positive. Schultz [9] reviews this work on the extinction phenomenon. Raymond [10] made 145 injections of normal serum of adults in ninety-seven cases of eruptive fevers and found that 82 per cent. of seventy-two frank scarlet fever cases gave positive reactions. Neuman [11] found that the phenomenon did not occur with foreign serum, distilled water or epinephrin in various high dilutions, and is enthusiastic enough to mention that the diagnostic results border on certainty.

Dorner [12] tested thirty-six cases of scarlet fever in which he got various results, obtaining 85 per cent. positive reactions when the serum was used from patients six weeks after the beginning of the disease. He also calls attention to the fact that when the rash was in decrescendo, results were less marked than when the rash was at its

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6. Hazelhorst, G.: Practical Applicability of Blanching in the Differential Diagnosis of Scarlet Fever, München. med. Wchnschr. **69**:116, 1922.

7. Mulsow, F. W.: Blanching of the Skin by Serum Injections in Scarlet Fever, J. Infect. Dis. **29**:557 (Nov.) 1921.

8. Rojo, D. J.: Extinction Signs in Scarlet Fever, Semana med. **1**:776 (May 11) 1922; abstr. J. A. M. A. **79**:594 (Aug. 12) 1922.

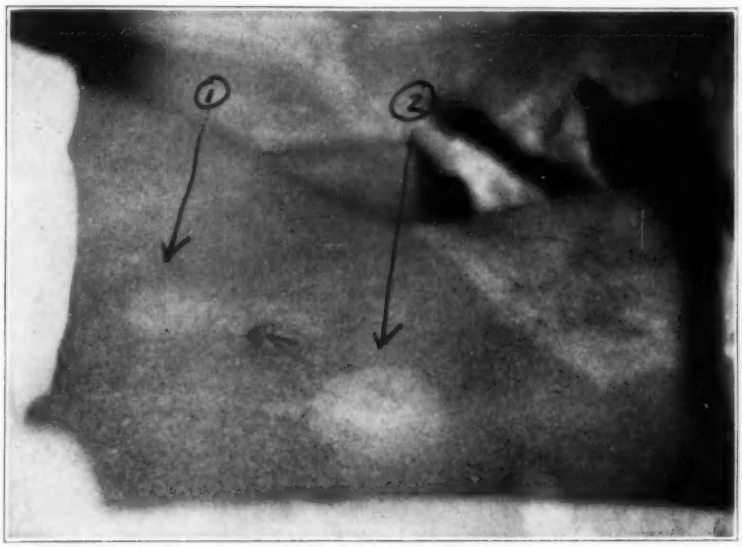
9. Schultz, W.: Induced Blanching of the Eruption in Scarlet Fever, Acta med. Scandinav. **54**:492 (April 6) 1921; abstr. J. A. M. A. **76**:1544 (May 28) 1921.

10. Raymond, H.: Differential Diagnosis of Scarlet Fever, Schweiz. med. Wchnschr. **51**:719 (Aug. 4) 1921; abstr. J. A. M. A. **77**:1054 (Sept. 24) 1921.

11. Neuman, J.: Induced Blanching of the Eruption in Scarlet Fever, Deutsch. med. Wchnschr. **46**:566 (Feb. 12) 1920; abstr. J. A. M. A. **75**:512 (Aug. 14) 1920.

12. Dorner, G.: Extinction Tests in Scarlet Fever, Med Klin. **17**:1543, 1921.

height. He noted that tetanus antitoxin, sheep and rabbit serum did not cause the reaction, and that serum from nurslings unlike any other serum does not blanch. Meyer-Estorf [13] found that the reaction was obtained best when the injection was made on the second day of the rash. Blum [14] injected sixty-four patients with 0.5 c.c. doses and tried to show that the degree of blanching is not dependent on the amount of the injection. He found that hemolysis destroyed the blanching power of the serum. He tested twenty-five cases of definite scarlet fever with varying results and in addition ten cases of surgical scarlet fever, in six of which there was a reaction although all ten were typically positive scarlet fever; in one of the latter, injection of normal



There were two definite responses in this patient to normal serum from different individuals and, in this instance, from different type groupings.

serum intradermally gave a negative reaction, i. e., no blanching, while the serum taken during the acute stage from this case when injected into another scarlet fever patient intradermally gave no blanching, i. e., a positive reaction. In another case, the blanching did not come on until forty-eight hours after the injection.

Marie [15] calls attention to the fact that injections of epinephrin in solution of 1:12,000,000 do not cause the discutient phenomenon.

13. Meyer-Estorf: Further Remarks Regarding Scarlet Fever Extinction, Med. Klin. **17**:526 (May 1) 1921.

14. Blum, J.: Extinction Phenomena in Scarlet Fever Diagnosis, München. Med. Wchnschr. **69**:466 (March 31) 1922.

15. Marie, P. L.: The Schultz-Charlton Phenomenon of Extinction and the Diagnosis of Scarlet Fever, Presse méd. **29**:1001, 1921.

Wöhlisch and von Mikulicz,[16] Salevsky[17] and Mouriquand[18] agree with the general findings of other authors.

As can be seen, there is great difference of opinion as regards the efficacy of the test, some authors claiming that it is specific, others that it is worthless. It occurred to us that further work should be done in order to evaluate this sign, as any procedure which would lead to a diagnosis in the borderline case of scarlet fever would be of distinct value.

We tested fourteen cases of definite scarlet fever at the height of the rash, using the following solutions: epinephrin 1:1,000; stock typhoid, triple vaccine; physiologic sodium chlorid solution; horse serum and serum from a convalescent case of about three weeks' duration. One-half cubic centimeter doses were used. Table 1 gives the results, positive indicating blanching; negative, no blanching.

TABLE 1.—*Results of Tests in Fourteen Cases*

	Epinephrin 1:1000	Typhoid Vaccine	Normal Serum	Horse Serum	Convalescent Scarlet Fever Serum
Positive	14	0	0	3*	3
Negative	0	14	14	11	11

* Questionable, but included in positive results.

TABLE 2.—*Results of Eight Tests*

	Positive	Negative
Normal human serum, 1 c.c.	7	1
Horse serum, 1 c.c.	0	8

Epinephrin produced blanching in all cases. The reaction, however, differs from that produced with human serum in that it begins in a few minutes and disappears in from five to six hours; the blanched area is much paler than normal skin, and is surrounded by a definite red margin from 3 to 5 cm. wide. Epinephrin was then injected in several diphtheria patients and the same result was obtained, lacking the contrast of the surrounding scarlet skin. The blanching obtained with human serum appears in from six to eight hours and persists until the rash fades. The area varies in size from 2 to 10 cm. in diameter, is round or oval, and may be definitely delimited from the surrounding erythematous skin, or may show an irregular indefinite margin. In some of the reactions, slender fingers of blanched skin extend into the rash even as far as from 8 to 10 cm.

16. Wöhlisch, E., and von Mikulicz Radecki: Skin Blanching Tests, Berl. klin. Wchnschr. **58**:389, 1921.

17. Salevsky: Discutient Phenomenon, M. J. No. 1, quoted in Zentralbl. f. d. Ges. Kinderh. **15**:33 (July 10) 1923.

18. Mouriquand: Scarlet Fever Signs, J. de méd. de Lyon **3**:66, 587, 1922.

Foreign protein in the form of stock typhoid vaccine and horse serum produced no typical blanching; although there were three instances of horse serum injection in which a rather indefinite disappearance of the rash occurred about 2 to 3 cm. in diameter. If these reactions were typical, a test which showed only three positive reactions out of fourteen experiments would be of little value. When we tested an additional eight cases of scarlet fever with horse serum and with convalescent scarlet fever serum, using a different serum than that used in the first experiment, we were surprised to find seven positive reactions out of eight as tabulated in Table 2, indicating that not all normal serums have the same power of blanching, a fact more evident from later experiments.

Normal serum was then compared with serum from a convalescing scarlet fever patient in the fourth week of the disease, and gave a higher percentage of positive reactions, as can be seen from Table 3.

TABLE 3.—*Results of Fourteen Tests*

	Positive	Negative
Normal human serum, 0.5 c.c.	9	5
Convalescent scarlet fever serum, 0.5 c.c.	3	11

TABLE 4.—*Results of Twenty-Seven Tests*

	Positive	Negative
Normal human serum, 1 c.c.	22	5
Convalescent scarlet fever serum, 1 c.c.	17	10

Most observers state that like reactions are obtained with serums in 0.5 c.c. and 1 c.c. amounts. To determine whether the amounts of serum influenced the results, twenty-seven cases of scarlet fever were tested, 1 c.c. each of normal and convalescent fever serum being employed. That a higher percentage of positive results can be obtained with the larger amount is shown in Table 4 as compared with the preceding table, consequently this amount was used in all subsequent experiments. Some observers have noted trauma when amounts larger than 0.5 c.c. are used, but we did not see in any case that the reaction was adversely influenced by injection of the larger amount.

An additional series of eight cases were then tested, normal and convalescent serum in 0.5 and 1 c.c. amounts being employed, with the following results:

TABLE 5.—*Results in Additional Series of Eight Cases*

	Positive	Negative
Normal human serum, 0.5 c.c.	4	4
Convalescent scarlet fever serum, 0.5 c.c.	0	8
Normal human serum, 1 c.c.	5	3
Convalescent scarlet fever serum, 1 c.c.	4	4

While few cases were tested in this manner, the results favored the larger amount for the test.

The following cases were tested by the intradermal injection of 1 c.c. of normal human serum: measles, six; diphtheria bacillus carriers with erythematous rashes, one; influenza with erythematous rash, two; urticaria, one; drug rash, one; serum rash, one; erythematous rash following administration of arsphenamin, one. There was no area of blanching in any instance. Two patients who had measles were injected with adrenalin, and blanching occurred in one.

Serum from definite scarlet fever cases during the first week of the disease was injected in eighteen cases, normal human serum being employed for control, with the following results:

TABLE 6.—*Results of Eighteen Tests*

	Positive	Negative
Normal human serum, 1 c.c.	18	0
Acute scarlet fever serum, 1 c.c.	0	18

Owing to the persistent negative results obtained when serum from a scarlet fever patient during the first eight to fourteen days of the disease is used, this test is of considerable value in establishing a diagnosis in doubtful cases. Serum from two patients, hospital orderlies who had been exposed to scarlet fever and who developed sore throats and were isolated as possible scarlet fever cases, was injected intradermally in a known case at the height of the rash. In both instances, blanching occurred, and the subsequent course confirmed the opinion that they were not developing scarlet fever. In another case, the serum of a nurse who was exposed in one of the scarlet fever wards and who developed a sore throat, nausea and vomiting associated with a slight erythema, was injected into a scarlet fever patient with a fully developed rash, and blanching was produced. The final diagnosis after twenty days' observation was acute streptococcic sore throat.

Serum from five cases of suspicious but not well defined scarlet fever was injected into the same number of cases of acute scarlet fever at the height of the rash, the reactions being negative in each case. Later, the diagnosis of scarlet fever was verified in these cases by the subsequent findings, extensive peeling, etc. The serum of two control cases of streptococcus sore throat was injected intradermally into two scarlet fever patients at the height of the rash, with negative results, i. e., with blanching. In one instance, the patient, a physician on service at the hospital, developed symptoms of a mild scarlet fever. We had on hand some of his blood serum which we had used in some other work, serum which had been withdrawn about four weeks prior

to his illness. This was used as a control in testing out the blanching power of serum withdrawn from him on the second day of his illness. The latter gave no reaction while the old serum caused definite blanching.

The observation has been made by others, and we have found it true in our cases, that serum from different healthy individuals produced different degrees of blanching. However, no explanation has been brought forth. To determine whether the iso-agglutinins and the hemolysins contained in the serum used and the blood of the patient had any influence on the degree of blanching, we obtained two sets of serums of the four types and tested fifteen patients with the first set and ten with the second. Jansky's classification is used in designating the different blood groups. The symbols (—) and (+) are used to express the degree of blanching.

TABLE 7.—*Results of Employment of Various Types of Serum on Different Types of Patients*

	Type of Serum Used for the Test			
Type of Patient	1	2	3	4
1	±	++	++++	+++
1	—	—	++++	+++
1	—	++	++++	+++
1	+	—	++++	—
2	—	++	+++	—
2	—	++	++++	++
2	—	++	++++	+++
2	+	++	++++	+++
2	±	±	+++	± (?)
2	++++	++	+++	+++
2	++	—	+++	—
3	+	±	+++	+++
3	++	+++	++++	++++
3	—	++	+++	++++
4	—	—	+	++

TABLE 8.—*Results of Employment of Various Types of Serums on Different Types of Patients*

	Type of Serum Used			
Type of Patient	1	2	3	4
1	—	+++	—	++
1	—	++	±	+
3	—	+++	±	++
3	+	+++	±	++
1	—	+	—	+
2	—	+++	—	++
1	—	++++	—	++
1	±	++	±	+
2	—	+++	—	++
1	+	++++	—	+

From results obtained, as shown in Table 7, it was seen that there were 100 per cent. positives in this series when the four types of serum were used. We also saw that Type 3 of this series caused the greater number of positive reactions in the greater degree; we also noted that if normal Type 1 serum of this experiment had been used alone there would have been only six definite reactions out of fifteen;

therefore, not all normal serums have the same blanching effect. We were interested to find out whether the second series of cases with various type serums would give the same number of positive reactions, especially with a new Type 3.

In the group findings presented in Table 8, Type 2 caused the greatest number of reactions.

A study of the foregoing tables discloses no relation between the degrees of blanching obtained and the blood groups of the patient and donor. It does, however, show that the serums from certain individuals persistently cause definite and marked blanching when injected intradermally in scarlet fever cases at the height of the rash, while those from other individuals produce blanching to a less degree or in some cases not at all.

A study of Tables 7 and 8 shows that the Types 2 and 3 used, caused the greatest number of reactions, Type 2 of Table 8 and Type 3 of Table 7. To see whether Type 2 of Table 8 and Type 3 of Table 7 would be positive, or produce blanching in the same individual, ten patients in the acute stage of scarlet fever were injected with Types 2 and 3 of Tables 7 and 8, with the following results:

TABLE 9.—*Results Obtained with Various Types of Serums*

Type of Serum Used for the Test			
Type 2 of Table 8	Type 2 of Table 7	Type 3 of Table 8	Type 3 of Table 7
±	+	+++	++
+	—	—	—
+	+	—	—
+	+	—	±
—	++	+++	—
±	+	++	—
—	±	+++	±
±	+++	+	—
—	++	++	±
±	+++	++	+

From the findings presented in Table 9, it can be seen that when a type serum is definitely positive, it is consistently positive, irrespective of the blood grouping of the patient or donor.

There have been as many theories as to the cause of this reaction as there have been men working on the problem. Meyer-Estorf [13] injected albumin serum and globulin serum intradermally and noticed no difference in the reaction; both caused blanching, but the number of cases studied was not impressive. He states that the exanthem is due to a hyperemia and that the extinction is due to a vascular constriction which Buschmann claimed that he had observed under the microscope. He assumes the presence of an antibody in the extinction serum, and points out the fact that animals which are immune to scarlet fever have no antibodies for scarlet fever, and when their serum is used for the test there is for that reason no blanching of the skin. Raymond,

quoted by Dorner,[12] thought that the vasoconstrictor action was due to epinephrin, but Dorner remarks that this could not be true since the points of blanching are not paler than normal skin, whereas epinephrin causes an extreme pallor. He, as well as Paschen,[3] believes that the scarlet fever serum has a vasodilatory action and that normal serum destroys this property. Dorner also calls attention to the significant fact that infant's serum does not cause any blanching. Hainiss, quoted by Blum,[14] stated that the normal serum lost its blanching power when rendered inactive. However, our experience, as well as that of others, would seem to indicate that the thermolabile factor had nothing to do with the reaction. Hemolysis destroys the blanching effect (Blum[14]). Marie states that the reaction is due to the albumins in the serum, the globulo-albumin and the seralbumin.

It has been considered by some that the blanching is due to a local immunity connected with the presence of antibodies in the normal serum. Schultz-Charlton states that the effect was not obtained from normal serum from which the proteins had been precipitated out by ferric-chlorid, and propounded the theory that the human serum contains inhibitory substances which act either on the vasodilatory principle of the scarlatinal virus or on the blood vessels directly. Wolf-Eisner, quoted by Meyer-Estorf,[13] seeks to explain the extinction phenomena along the lines of biologic immunity. He proceeds from Pirquet's theory of the cause of scarlet fever, which attributes the cutaneous manifestations of the disease to a meeting of antibodies in the skin, and relates the phenomenon to other immunity reactions as that of the tuberculins. No theory brought forth as yet definitely explains the phenomenon in question. What this substance is is not known, but its importance warrants further study.

SUMMARY

One hundred and thirty-three scarlet fever cases were studied and compared with twenty-two control cases in an attempt to evaluate the extinction phenomenon of Schultz-Charlton. We were able

1. To confirm the fact that injections of serum from acute cases of scarlet fever, injected intradermally in another case of acute scarlet fever in the exanthematous stage will not cause blanching.

2. To show that not all serums are similarly potent in producing blanching, few normal serums producing no blanching at all, a fact which limits the application of this test in its negative phase.

3. To show that the injection of normal serum in cases with rashes other than the scarlet fever rash does not cause the reaction.

4. To show that normal serums give a greater number of reactions than convalescent scarlet fever serum.

5. To show that normal serums in 1 c.c. amounts give a greater number of positive reactions than normal serums in 0.5 c.c. doses.

6. To point out that if the serum is used and produces a definite reaction in one case, it will produce the same reaction in nearly 70 per cent. of other cases tried.

7. To point out that if two serums are used which produce definite blanching in a given case, they will complement each other and will give 100 per cent. reactions in other cases tried.

CONCLUSIONS

1. There is present in the blood of some normal and some convalescent scarlet fever serums a substance which, when injected intradermally, will cause the rash of scarlet fever to disappear at the seat of the injection, as first pointed out by Schultz-Charlton.

2. The serum from an acutely ill scarlet fever patient in the erythematous stage will in the great majority of cases not cause a disappearance of the rash when injected intradermally into another acutely ill scarlet fever patient in the erythematous stage.

3. The cause of this reaction is not known.

ENTEROLITH AS CAUSE OF CHRONIC OBSTIPATION IN A BREAST-FED INFANT *

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AND

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True intestinal stone in early life is rare. We were unable to find any reported in a child under 6 years of age. The case to be described occurred in an infant exclusively breast-fed, who suffered complete obstruction, and was operated on, at the age of 8 months.

REPORT OF CASE

History.—W. J., a boy, aged 8 months, had been fairly well nourished on the breast, with a monthly gain of 1 pound (453.6 gm.). He had never had an unassisted bowel movement. Meconium was evacuated for first two weeks by rectal irrigation, and fecal matter was not seen in the stool until after the infant was 2 weeks old, when hard fecal masses began to be obtained by irrigation. When castor oil was given, the stools would be liquid, and on one occasion blood was present in stool. During the eight months of the obstinate constipation, numerous laxatives, including croton oil, were tried by the attendant, often unsuccessfully for several days at a time. At no time was magnesia given. Diarrhea resulted on two occasions, between the ages of 6 and 7 months. The only other complications were eczema for a few weeks in the early months, and abdominal distension most of the time.

At 8 months of age, the infant developed fever (2 degrees) and the appetite failed, but he took water eagerly. Vomiting occurred twice daily. On the third day of illness, May 31, 1923, the patient came under our observation at St. Luke's Hospital.

Examination.—At this time, the weight was 17 pounds (7.7 kg.). The infant had an anxious expression, and the eyes were somewhat sunken. The temperature 102. The abdomen was markedly distended, so that palpation was difficult. No roentgenogram was taken before barium sulphate was administered. This was unfortunate, as it would have been of value to learn whether a shadow was cast by the intestinal mass independently of the barium sulphate. However, a barium sulphate enema did not pass the sigmoid flexure; a barium sulphate meal required forty-eight hours to traverse the intestinal tract, and there was barium sulphate still present in the colon and sigmoid at the end of 120 hours. In the middle portion of the sigmoid, an area of 1½ inches was less opaque than the surrounding structures. Under anesthesia, a hard globular mobile mass was easily felt in the region of the umbilicus and diagnosed as an enterolith.

Preoperative Laboratory Findings.—Examination of the blood revealed: leukocytes, 23,200; polymorphonuclears, 47; large lymphocytes, 5; small lymphocytes, 47; transitionals, 1.

In the urine, there was a trace of bile and an occasional pus cell, but no blood. A few granular casts and cylindroids, and a number of uric acid crystals were noted, but no indol was present.

Operation (E. L. M.).—A right rectus incision, 1½ inches above and 2½ inches below the umbilicus was made, with delivery of a redundant loop of sigmoid much enlarged in length and in caliber. The mass could be moved

* Received for publication, Oct. 31, 1923.

slightly in both directions within the bowel. A longitudinal incision was made at the lower end of the bowel and a stony mass was expressed. The intestinal wall was thickened. Closure of the sigmoid and abdominal opening was without incident.

Course.—The infant had a normal convalescence, with the exception of the need for assistance in bowel evacuation, for which suppositories were used.

Five days after operation, the blood showed a relative and actual increase in the neutrophiles: white cells, 18,200; polymorphonuclears, 79 per cent.; large lymphocytes, 2 per cent.; small lymphocytes, 19 per cent.

The wound healed normally, and no elevation of temperature was present after this time. The infant was taken from the hospital one week after the operation.

Specimen.—The stone was oval, 3½ by 5 cm.; weighed 40 gm., and was dark gray and smooth. When cut through, the outer portion was laminated, stony, unyielding, the center was less dense, smooth and gray, and contained no nucleus of any different composition. When kept in specimen jar solution for several months, the exterior of the stone eventually softened and crumbled into the solution, forming a dirty grayish opaque mixture. Analysis [1] of the stone showed that the ash consisted principally of calcium (50.51 per cent.), magnesia (3.19 per cent.), and phosphorus (18.44 per cent).

Outcome.—Two months after the removal of the stone, the infant had gained 2½ pounds (1,134 gm.). The bowels had not moved voluntarily, and a mild laxative or an occasional suppository was required. The child was still on the breast, and weaning was advised, so as to provide food with more residue.

OCCURRENCE

Inspissated and impacted meconium as a cause for fetal or newborn obstruction has been reported by Bullowa and Brennan.[2] Their case, in an infant 2 days old, came to necropsy, and the obstructive mass was found to consist of white, inspissated, impacted and stratified meconium, evidently of intrauterine origin.

Fecal impaction is of more common occurrence. It is probable that, in most cases, the mass remains a fecalith, or that a nucleus of a foreign body gains accretions from the feces or from the mineral excretions of the intestinal mucosa. Dorning reported a fecal impaction in a 2-year-old child due to the swallowing of particles of cloth, and he believes that insoluble mineral substances, such as magnesia or bismuth, when taken over a long period may accumulate and form an obstruction.[3]

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1. Analysis was made by Drs. Walter M. and Roy Cross. The hypothetical combination of the ash was as follows: Calcium phosphate ($Ca_3 PO_4)_2$, 40.35 per cent.; calcium carbonate ($Ca Co_3$), 51.33 per cent.; magnesium carbonate ($Mg CO_3$), 6.83 per cent. The organic matter other than fat and cholesterol most probably existed as fatty acids combined with the lime and magnesia of the ash as lime and magnesium soaps.

2. Bullowa, J. G. M., and Brennan, R. E.: Intra-Uterine Intestinal Obstruction from Inspissated and Impacted Meconium, J. A. M. A. **73**: 1882 (Dec. 20) 1919.

3. Dorning, John: Intestinal Obstruction in Early Life, Med. Rec. **45**: 556, 1894.

Shattock states that there are only three specimens of intestinal calculus in the human collection at the Royal College, London. A few instances are recorded in which the obstinate constipation of adults, which had existed since childhood, was due to an enterolith. Simpson [4] reported an enterolith in a 16-year-old girl in Scotland, who, as a child,

Fig. 1.—Enterolith in sigmoid flexure. The oval mass is within the dotted line.

had been in the habit of chewing heather while herding cattle. The enterolith was 4½ by 2¾ inches (11.3 by 6.9 cm.), and was composed of heather roots and twigs compressed and surrounded by mineral accretions of stony hardness. The mass was in the ileum, 16 inches

4. Simpson, B. S.: Edinburgh M. J. **30**: 176 (April) 1923.

above the ileocecal valve. It is interesting that stones, avenoliths, formed from ingested oatmeal, were noted in Scotland.[5]

Boardman [6] described his case, in an adult, as beginning in infancy while the child was being fed on the breast.

Enteroliths are not mentioned in acute intestinal obstruction in young children. Peterson [7] had fifty-five such cases, mostly in infants, one of which was due to a foreign body, not an enterolith. Gant [8] has collected fifty-four cases, the three youngest patients being between 6 and 8 years.

Fig. 2.—Sigmoid, showing distension by stone, at time of operation.

Treves ("Intestinal Obstruction") divides enteroliths as to composition into (1) concretions formed of phosphate of lime or magnesia, or of triple phosphates or of mixtures of these salts; (2) "oat-stones" of low specific gravity composed mostly of vegetable matter, fecal matter and calcareous material, and (3) concretions formed of insoluble mineral matter which has been swallowed as medicine, mostly magnesia.

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5. In a personal communication, Leonard Findlay, Glasgow, said that he has not known of an enterolith in children in Scotland.

6. Boardman, W. W.: Am. J. Roentgenol. **10**:369 (May) 1923.

7. Peterson, E. W.: Surg., Gynec. & Obst. **35**:436 (Oct. 22) 1923.

8. Gant, S. G.: Diseases of Rectum, Anus and Colon, Philadelphia, W. B. Saunders Company **2**: 494, 1923.

COMMENT

Formation.—While stones may develop in early life, there is a history of long-standing constipation and many years elapse [9] before the stone reaches the complete obstructive size or situation, in practically all cases in adult life. In the case here reported, no magnesia, no bismuth and, until the time of operation, no barium had ever been administered. There was a history of difficult evacuation of the bowel since birth, and a prolonged period of meconium stools, with no fecal matter appearing for two weeks. There had been no jaundice at any time. The nucleus had evidently been formed during intrauterine life or shortly afterward, and contained no foreign body. Inspissated meconium, dehydrated feces and accretions from the secretions of the intestinal mucosa offer, it seems to us, the only explanation of intestinal calculus in our case. The possibility of the origin in a diverticulum is of theoretical interest.

Mechanism.—The mechanism of stone formation and retention is interesting, and is not entirely understood. Young children swallow many indigestible bodies of various sizes, sometimes exceeding the usual circumference of the intestine. Such foreign bodies are usually passed finally, or may remain for some time in the bowel, usually the rectum, without mineral accretions. It is difficult to understand why peristalsis, which may even become increased by the presence of the stone, does not pass the mass while it is yet small.[10] Fecal matter will be pushed past the stone without dislodging or evacuating it. In the reported cases, most stones are found in the rectum. In our case, the stone was found in a favorable location—a flexure, where, by its ball-valve action, liquid feces were at times permitted to pass.

Diagnosis.—Obstinate constipation is of first importance and is the cause for seeking medical advice. The general symptoms are likely at times to be stormy, and are sufficient to suggest an acute obstruction: vomiting, periods of distress and anxiety, tenderness, colic, distension and flatulence, peristaltic wave and diarrhea with mucus, blood or pus. So rare is the intestinal stone and so difficult is palpation of the mass that stone is not considered until the individual comes to operation, or a roentgenographic examination is made. Under anesthesia, it was possible in our case to feel the tumor through a relaxed abdominal wall, but the roentgenogram is essential for differentiation. As has been suggested by Pfahler,[11] the stone, if of sufficient density, should first be sought by a roentgenogram without a barium sulphate coating, and, if this procedure is inconclusive, the opaque meal will outline the mass, which of itself may be of insufficient density to cast a shadow.

9. Cowan, J. F.: Enteroliths, S. Clinics of N. Amer. **2**:401 (April) 1922.
10. Boardman, W. W.: Am. J. Roentgenol. **10**:369 (May) 1923.
11. Pfahler, G. E.: Diagnosis of Enteroliths, Surg. Gynec. & Obst. **21**:14. 1915.

REPORT OF A CASE OF HEALED MILIARY TUBERCULOSIS *

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FREDERICK, MD.

The pathologic examination in a case of generalized miliary tuberculosis in childhood shows why this disease is nearly always fatal. There is a softened focus of tuberculosis, usually in a lymph node, which has discharged into a blood vessel, not once, but several times, the cavity having undoubtedly filled. Evidence of repeated infection is found in the size of the resultant tubercles. There are crops of different sizes, but the tubercles that belong to one crop are of approximately the same size. This indicates an eruption of tubercle bacilli into the blood stream at different intervals. The discharge of tubercle bacilli may be so overwhelming and produce so extensive and generalized an infection as to prove fatal in a short time. But if it does not, successive discharges of tubercle bacilli can hardly be borne; and, from the nature of the lesion in the tuberculosis focus, successive discharges are the nearly invariable rule.

It is conceivable that the focus of dissemination may be so small that the number of tubercle bacilli discharged may not produce a severe infection, and healing of the focus, after it has been evacuated, may rapidly take place. Under such conditions, the infection, while generalized, may not be sufficient to overwhelm the organism, and recovery may occur. I wish to report what appears to be an example of healed miliary tuberculosis.

REPORT OF CASE

History.—N. D., a white boy, aged 11 years, admitted to the Harriet Lane Home, Oct. 21, 1919, the youngest of four children, all the others being well, and whose father had had asthma for many years, was breast fed for a year and developed normally. At 1 year of age, he had measles followed by "catarrhal croup," during which he had difficulty in getting his breath. Following this, he had attacks of shortness of breath, at first every month or two, and continuing up to the present time, though at rather longer intervals. These attacks were brought on by cold, excitement or violent exertion, or when the patient was in a room where there was much tobacco smoke. The adenoids and tonsils were removed at the age of 5. At 8 years of age, the boy had pertussis; at the age of 10, influenza followed by pneumonia. He was brought to the hospital because of the asthmatic attacks, and remained for five days. The temperature during this time was normal.

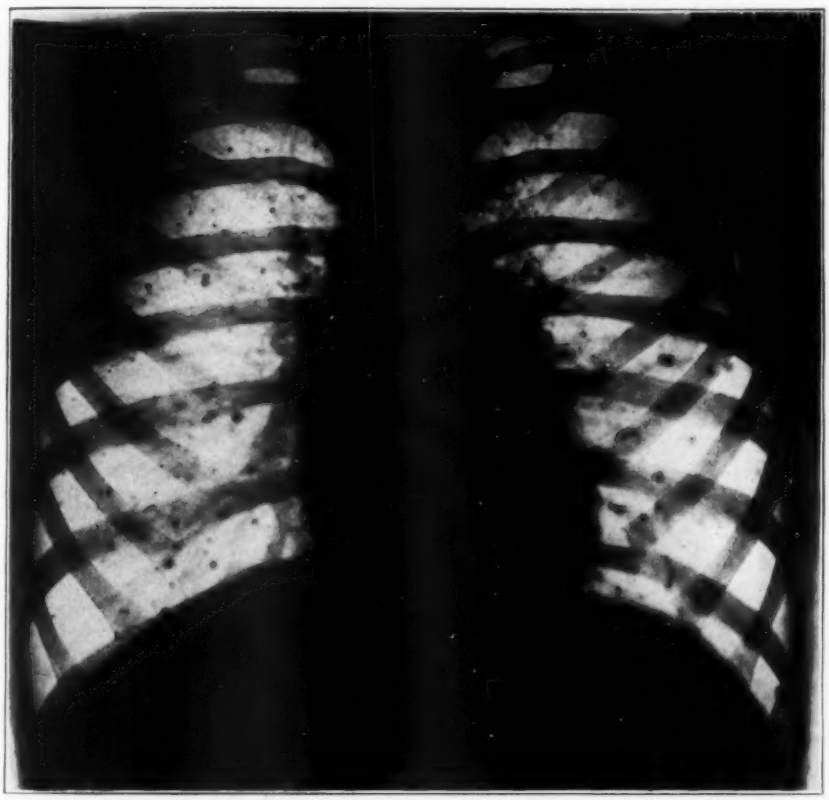
Physical Examination.—The boy was moderately well developed, somewhat underweight, weighing 59 pounds (26.8 kg.). The lungs on percussion were normal. On auscultation, a few scattered moist râles were heard. The heart

* Received for publication, Sept. 26, 1923.

* From the Department of Pediatrics, Johns Hopkins University.

was normal. An intracutaneous injection of tuberculin, 0.1 mg., produced no reaction. A roentgenogram of the chest revealed small opaque areas scattered throughout both lungs even to the margins. There was no evidence of an accumulation of these in the vicinity of the roots of the lungs. Stereoscopic plates showed that the nodules were situated in the lung substance. The nodules were, roughly, uniform in size and appeared to be small areas of calcification. Roentgenograms of other portions of the body showed nothing similar.

Course.—The boy has been under observation for nearly four years. His health has remained good. His asthmatic attacks are infrequent. Several recent



Appearance of chest, showing calcified nodules scattered through both lungs.

roentgenograms show exactly the same picture as did the first. Subsequent tuberculin tests were made. There was a definite reaction on injection of 5 mg. of tuberculin intracutaneously.

The wide and even distribution of the nodules point to a dissemination by the blood stream rather than by the bronchi or lymphatics. The possibility of metastases of some primary undiscovered tumor has been eliminated by the length of time that has elapsed. It seems that the presence of several nodules, presumably calcified, can be accounted for most satisfactorily by assuming that the boy at some time, following

measles or pneumonia, had an eruption of tubercle bacilli into his blood that caused a number of isolated lesions; but the infection was overcome, as there was no recurrence. Subsequently, calcification of the lesions took place. Miliary tuberculosis in adults, running a chronic course or unsuspected until detected by roentgenograms, has been reported recently by Bierman [1] and by Stivelman and Hennell.[2] I have seen an example of the form described by the latter authors. Unsuspected and healed miliary tuberculosis in a boy of 14 years, with death occurring from quite a different cause, has been reported by Opie and Andersen.[3] The roentgenograms taken postmortem of the lungs of this child are strikingly similar to those of my patient. This fact, taken in connection with the positive tuberculin reaction, renders the diagnosis reasonably certain.

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1. Bierman, M. I.: Minnesota Med. **5**:661-665 (Nov.) 1922.
2. Stivelman, B. P., and Hennell, Herman: J. A. M. A. **80**:536 (Feb. 24) 1923.
3. Opie, E. L., and Andersen, H.: Am. Rev. Tuberc. **4**:629 (Nov.) 1920.

TUBERCULOSIS OF THE TONSILS AND ADENOIDS

A CLINICAL AND ROENTGEN-RAY STUDY OF FIFTY CASES OBSERVED FOR FIVE YEARS AFTER OPERATION *

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Since 1912, every tonsil and adenoid removed at operation in the Johns Hopkins Hospital has been studied histologically. While no attempt is made to examine the entire tonsil or adenoid by serial sections, the blocks selected for microscopic section and the number of slides obtained for study are uniform throughout the whole series of 3,260 cases. This paper deals entirely with those cases in which a tuberculous lesion was discovered in the tonsil or adenoid; other pathologic conditions of a nontuberculous nature that were noted in this study will be presented in a subsequent publication.

Virchow,[1] in 1864, made the statement that tuberculosis of the tonsil is a rare occurrence, and that this tissue is extraordinarily immune to tuberculous infection. For a period of fifteen years following Virchow's publication, no experimental or pathologic observations were made on tuberculosis of the lymphoid tissue in the pharynx, with the exception of an occasional note on the ulcerative lesions in this region that occur in the late stages of pulmonary tuberculosis. During the course of some experimental observations on the effect of feeding tuberculous meat, Orth,[2] in 1879, observed ulcerated tuberculous lesions in the tonsils. Baumgärten [3] repeated the feeding experiments of Orth five years later, and noted, in addition to the lesions in the tonsils, a tuberculous cervical adenitis. He commented on the resemblance of the condition in these glands to scrofula, but was confused because a tuber-

* Received for publication, Nov. 6, 1923.

* From the Department of Surgery of the Johns Hopkins University and Hospital.

* Many of these references were made possible through the aid of the Surgeon-General's Library.

1. Virchow, R.: Die krankhaften Geschwülste **2**:674, 1864.

2. Orth, J.: Experim. Untersuchungen über Fütterungstuberculose, Virchows. Arch. f. path. Anat. **76**:217, 1879.

3. Baumgärten: Ueber die Uebertragbarkeit der Tuberkulose durch die Nährung und über Abschwächung der pathogenen Wirkung der Tuberkelbacillen durch Faülnis, Centralbl. f. d. med. Wissensch. **22**:347, 1884.

culous cervical adenitis in man is found so rarely in association with an advanced pulmonary lesion.

Lermoyez,[4] Cornil [5] and Abraham [6] were among the first to present clinical cases of tuberculous tonsils and adenoids removed at operation. Lermoyez, in 1894, removed the adenoids in a child, aged 6 years, who

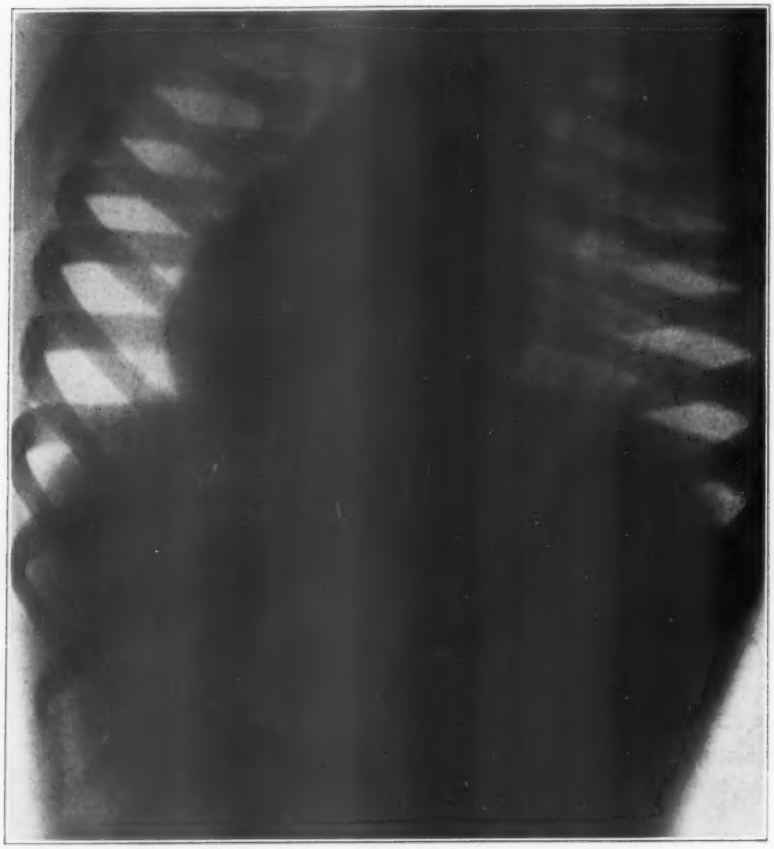

Fig. 1.—J. H., colored boy, aged 4, two and a half years after operation. The lungs were clear. The mediastinal glands were enlarged, the condition suggesting mediastinal tuberculosis. At the time the roentgenogram was taken the child was admitted to the hospital for acute nephritis. Aside from edema, the physical examination was negative. Both tonsils and adenoids and the cervical glands on the left side were found to be tuberculous two and one half years ago.

4. Lermoyez, Marcel: Des végétations adénoides tuberculeuses du pharynx nasal, Bull. et mém. Soc. méd. d. hôp. de Paris **11**:559, 1894.

5. Cornil, V.: Sur l'angine tuberculeuse, J. d. conn. méd. prat. **42**:193, 1875.

6. Abraham, P. S. Tubercle of the Tonsils, J. Acad. Med. Ireland **3**:358, 1885.

had no clinical evidence of tuberculosis in the lungs or elsewhere in the body. He found tubercles in the adenoids and was able to demonstrate tubercle bacilli in the lesion.

During the period from 1885 to 1900, Orth,[7] Krückmann,[8] Schlänker,[9] Ruge[10] and Friedmann[11] reported postmortem examina-

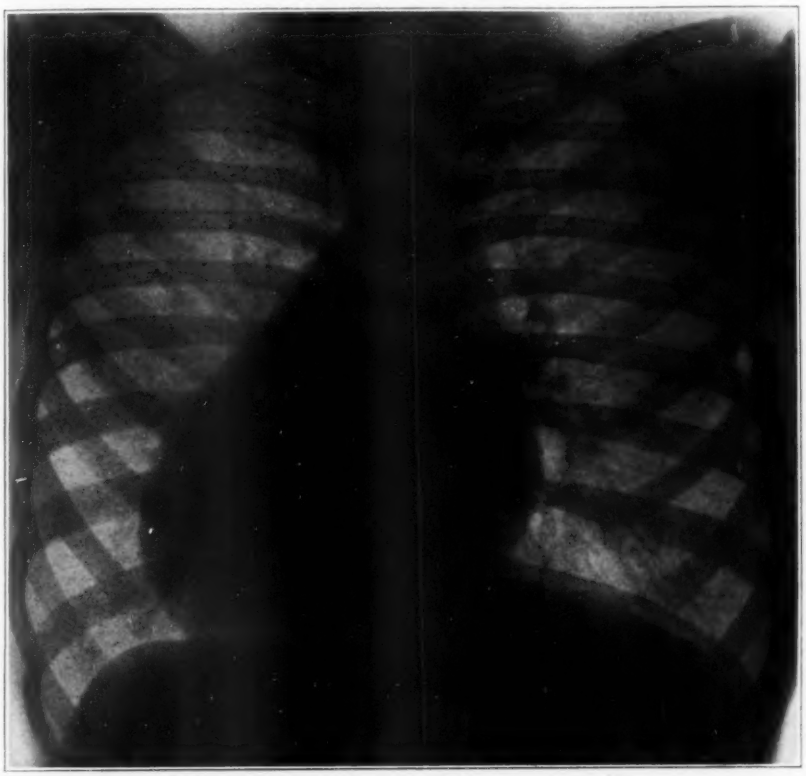

Fig. 2 (J. H.).—Appearance of chest of patient two years later. The lungs were still clear. There was a decided reduction in size of the mediastinum. The physical examination was negative. The cervical adenitis cleared up within a year after the operation. The child received tuberculin treatment for one year after operation.

7. Orth, J.: Lehrbuch der spec. patholog. Anat., Berl., 1889, Ib, 663.

8. Krückmann, E.: Ueber die Beziehungen der Tuberkulose der Halslymphdrüsen zu der Tonsillen, Virch. Arch. f. path. Anat. **134**:534, 1893.

9. Schlänker, E.: Untersuchungen über die Entstehung der Tuberkulose der Halsdrüsen, besonders über ihre Beziehungen zur Tuberkulose der Tonsillen, Virchows. Arch. f. path. Anat. **134**:161, 1893.

10. Ruge, H.: Die Tuberkulose der Tonsillen vom klin. Standpunkte, Virchows Arch. f. path. Anat. **144**:431, 1896.

11. Friedmann, F.: Ueber die Bedeutung der Gaumentonsillen von jungen Kindern als Eingangspforte für die tuberkulöse Infection, Beitr. z. path. Anat. u. z. allg. Path. **28**:66, 1900.

tions in which tuberculous lesions were found in the tonsils. In Friedmann's cases, no tuberculosis was found elsewhere in the body; in the others, the cervical glands were involved, but the microscopic appearance indicated that the tonsillar lesion was the most advanced and apparently the primary portal of entry. Dmockowski,[12] in 1891, experimentally infected the tonsils and cervical glands, and demonstrated, in

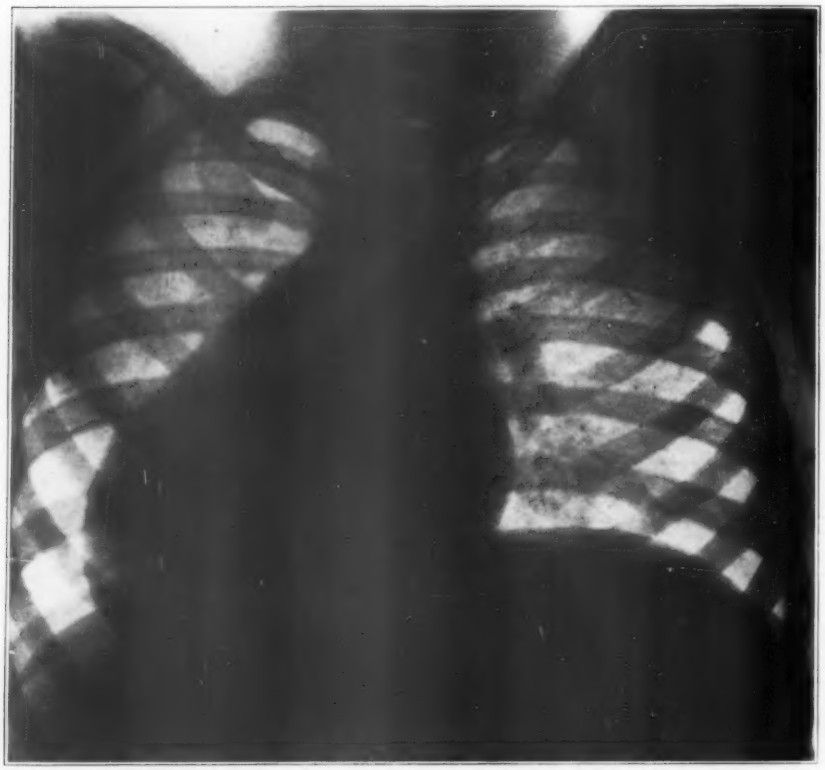

Fig. 3 (J. H.).—Five years after operation, the lungs were clear. The mediastinum was now of normal size. The heart was enlarged. The patient appeared well nourished. He had been free of any symptoms except shortness of breath. Physical examination showed a mitral insufficiency, but nothing abnormal in the lungs.

microscopical sections, the tubercle bacillus in the tonsils, the cervical glands and the intervening lymph channels. Dieulafoy,[13] in 1895,

12. Dmockowski, Z.: Ueber secundäre Erkrankungen der Mandeln und der Talgdrüsen bei Schwindsüchtigen, Beitr. z. path. Anat. u. z. allg. Path. **10**:481, 1891.

13. Dieulafoy, M.: Tuberculose larvée des trois amygdales, Bull. de l'Acad. de méd., Paris **33**:437, 473, 1895 (discussion by Cornil, p. 498).

inoculated animals with bits of tonsillar tissue that had been removed at operation from individuals with no clinical evidence of tuberculosis. He found that eight of the sixty-one animals so inoculated developed tuberculosis; indicating that 13 per cent. of these sixty-one patients harbored tubercle bacilli in the tonsils, but gave no clinical evidence of tuberculosis locally or elsewhere in the body. The inoculation experi-

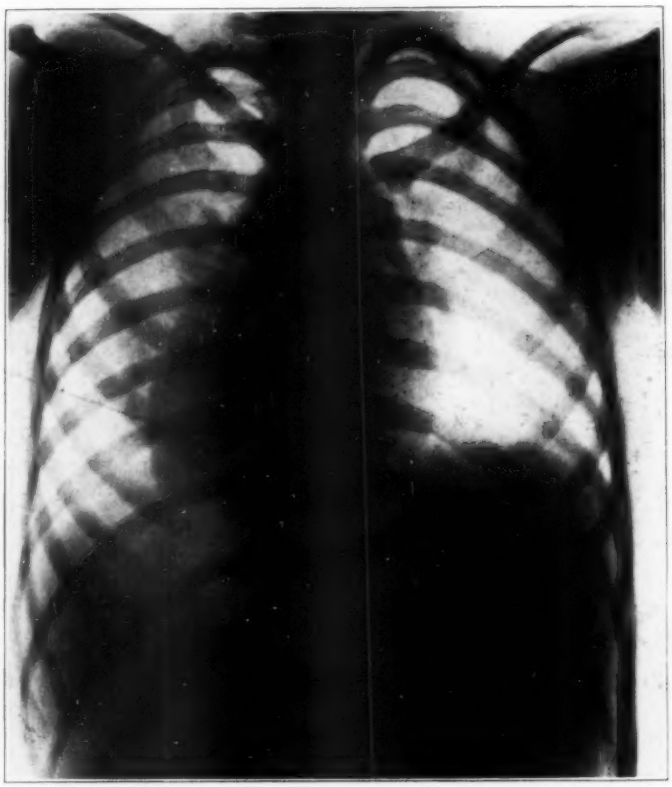

Fig. 4.—C. N., white girl, aged 7, two months after operation. The lungs were clear. The mediastinal glands were enlarged, suggesting mediastinal tuberculosis. Physical examination before operation was negative except for tuberculous cervical adenitis. Both tonsils were found to be tuberculous.

ments with adenoid tissue showed that 20 per cent. of the thirty-five individuals examined had tubercle bacilli in this region. This report was violently criticized by Cornil, but aroused interest in the question as to whether or not the lymphoid tissue in the pharynx is a common portal of entry in tuberculosis.

The following publications that have appeared during the past twenty years are important. Harbitz,[14] in 1905, found that cervical lymph glands removed soon after death from individuals killed in accidents often contained tubercle bacilli, virulent for animals, although no gross or microscopic evidence of tuberculosis could be demonstrated in the glands. It is his impression that organisms of all kinds are constantly passing through the lymphoid tissue in the pharynx to

Fig. 5 (C. N.).—Four years later, the mediastinum was almost within normal limits, but there was increased root fibrosis with thickened bronchial shadows extending into the lung proper of both upper lobes. Symptoms of cough, fever and gastro-intestinal disturbances developed six months after operation. The breath sounds were harsh, but no râles were heard. The patient received tuberculin treatment and very careful general hygienic care and has had no symptoms for the past two years.

the cervical glands. In many instances, these organisms cause no local or general symptoms until some intercurrent infection lowers the

14. Harbitz, F.: Studies in the Frequency, Localization and Modes of Dissemination of Tuberculosis, with Special Reference to Its Occurrence in the Lymph Nodes and During Childhood, J. Infect. Dis. **2**:143, 1905.

resistance. Wood,[15] in Philadelphia, whose anatomic studies on the lymphatic drainage of the tonsils are well known, has made important contributions in regard to tuberculous and other infections of the tonsils. Wood describes lymphatics from the tonsils that lead directly to a cervical lymph gland located in the triangle between the ascending process of the mandible, the anterior border of the sternomastoid and the posterior belly of the digastric muscle. He calls this the tonsillar

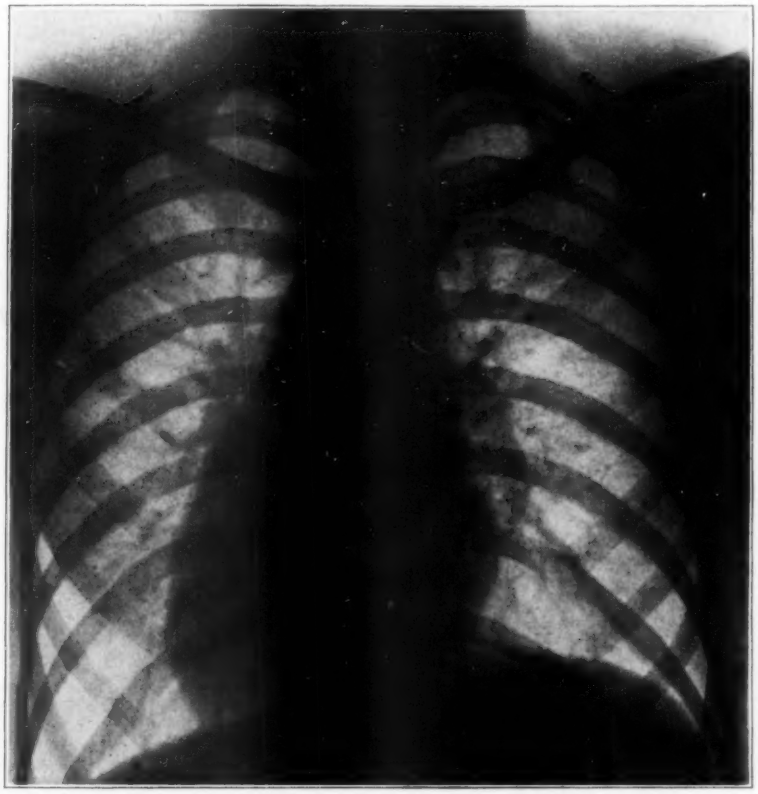

Fig. 6 (C. N.).—Seven years after operation, the mediastinum was normal. There were definite changes in both uppers beneath the clavicles, more marked on the right side. The apices were clear. The condition suggested an old fibroid tuberculosis. The percussion note was impaired and the breath sounds were roughened over both uppers. The patient has been free of symptoms for over five years, and is able to keep up her school work.

gland. It is usually the first of the cervical glands to enlarge when the tonsil is acutely infected; it is almost invariably enlarged in association

15. Wood, G. B.: The Significance of Tuberculous Deposits in the Tonsils, J. A. M. A. **44**:1425 (May 6) 1905; Lymphatic Drainage of the Faucial Tonsils, Am. J. M. Sc. **130**:216, 1905.

with a chronic tonsillitis, and in both tuberculous cervical adenitis and Hodgkin's disease a visible increase in size of the glands near the angle of the jaw is often the earliest evidence of the disease. Van Ree,[16] in a recent study of 205 patients with tuberculous cervical adenitis, found the oldest lesion in the tonsillar gland in 50 per cent. of the cases. Mitchell,[17] in 1914 and 1916, published several interesting papers on the frequency and the types of tubercle bacilli in the milk in England,

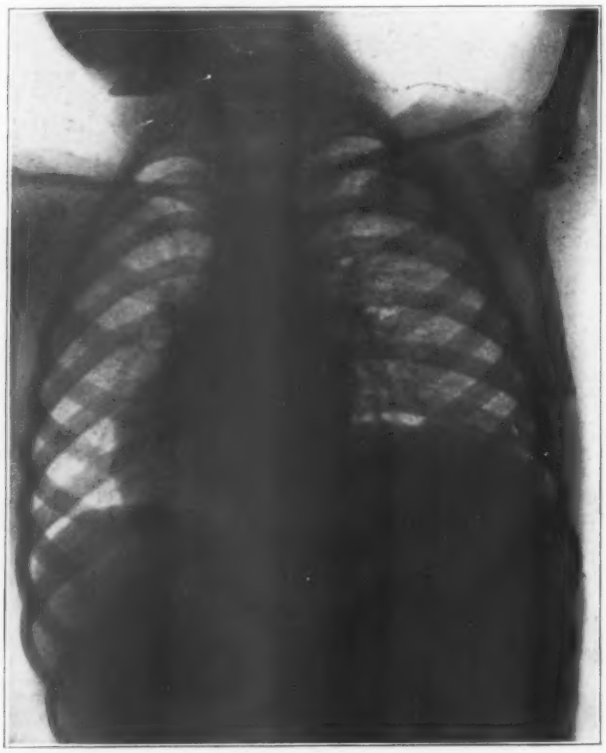

Fig. 7.—E. B., white girl, aged 3; before operation. The mediastinal glands were not enlarged. The symmetrical bilateral fibrosis indicated a nontuberculous infection. Physical examination was negative except for phlyctenular conjunctivitis and chronic tonsillitis. After operation, both tonsils and adenoids were found to be tuberculous. Tuberculous cervical adenitis developed on the left side within a month after the operation. The gland was incised.

and on primary tuberculosis of the faucial tonsils in children. He finds the bovine bacillus in 75 per cent. of the tuberculous tonsils in children

16. Van Ree, A.: Die tuberkulose der amandelen, Nederl. Tijdschr. v. Geneesk. **12**:796, 1919.

17. Mitchell, A. P.: Primary Tuberculosis of the Faucial Tonsils in Children, J. Path. & Bacteriol. **21**:248 (April) 1917; Report on the Infection of Children with the Bovine Tubercle Bacillus, Brit. M. J. **1**:125, 1914.

under 10 years of age. Park and Krumweide [18] of the New York Board of Health arrived at much the same conclusion in regard to the type of organism in tuberculous cervical adenitis in children. Only one or two cases of pulmonary tuberculosis have been reported in which the bovine bacillus was the organism responsible for the lesion, but there are numerous studies that show the frequent association of tubercles in the tonsils or adenoids and tuberculous glands of the neck.

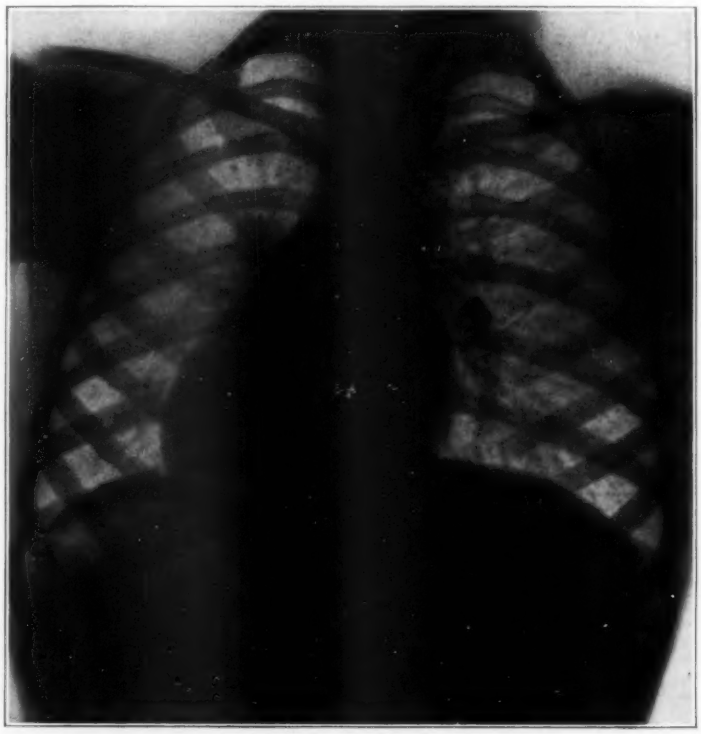

Fig. 8 (E. B.).—Three years later, the lungs were clear, the mediastinal glands slightly enlarged. The tuberculous sinus on left side of the neck healed within three months. The patient has never had any symptoms of tuberculosis and appears in good health. The physical examination was negative.

Enlarged glands of the neck are rarely seen in the sanatoriums in which advanced cases of pulmonary tuberculosis are treated.

It would seem justifiable to conclude, therefore, that in a majority of the cases of the tuberculous tonsils and cervical glands in children, the condition is primarily due to the drinking of milk from infected

18. Park, W. H., and Krumweide, C.: Relative Importance of the Bovine and Human Types of Tubercle Bacilli in the Different Forms of Human Tuberculosis, J. Med. Research **18**:205, 1910.

cows. In our series of 138 cases of tuberculosis of the tonsils or adenoids, we found a group of fifteen children in whom the adenoids alone were involved. In each of these fifteen children, repeated physical and roentgen-ray examinations have failed to disclose any evidence of tuberculosis in the cervical or mediastinal glands or in the lungs, although the tuberculin test remains positive. It is difficult to explain this apparently primary infection in the naso-pharynx on any other basis than that of an air-borne infection. Our methods, however,

TABLE 1.—*Summary of Finding of Various Authors*

Authors	Year	Total Number	Number Tuberculous	Percentage	Remarks
Tonsillectomy and Adenoidectomy in Patients Showing No Clinical Tuberculosis					
Friedmann	1900	53	1	1.9	
Wood	1905	50	1	2.0	
Bandilier	1906	78	1	1.3	Mostly adults
Mathews	1910	57	0	0.0	
Sewall	1911	772	30	3.9	Includes eleven patients with cervical adenitis
Barnes	1914	159	3	2.0	
Willis	1914	108	9	8.3	
Mitchell	1916	100	9	9.0	
Crowe	1917	1,000	46	4.6	Includes twenty-three patients with cervical adenitis
Van Ree	1917	92	2	2.2	Tonsillectomy only
		2,460	102	4.15	
Tonsillectomy and Adenoidectomy in Patients with Tuberculous Cervical Adenitis					
Carmichael	1910	50	7	14.0	
Mitchell	1916	106	41	38.7	
		156	48	30.76	
Adenoidectomy in Patients with No Clinical Tuberculosis					
Brindel	1896	64	8	12.5	
McBride	1897	100	3	3.0	
Luzzatti	1897	50	2	4.0	
Brieger	1898	75	5	6.7	
Lewin	1899	200	9	4.5	
Pfiffl	1899	100	3	3.0	
Wex	1899	210	7	3.4	
Rethi	1900	100	6	6.0	
Hynitsche	1900	180	7	3.9	
Lartigau	1902	75	10	13.4	
Van Ree	1919	62	7	11.3	
		1,216	67	5.5	

are subject to a large percentage of error, and no attempt was made in our series to determine the type of the tubercle bacillus, so we are unable to say definitely that the tonsils were not involved or that the organism was of the human type.

Table 1 presents a summary of all reports, of at least fifty cases, in which the tonsils and adenoids were removed at operation from patients with no clinical evidence of pulmonary tuberculosis.

In a previous publication, in 1917, it was pointed out that a routine histologic examination of 1,000 tonsils and adenoids removed at operation showed that forty-six of these 1,000 patients had a tuberculous infection of the tonsils or of the lymph-adenoid tissue removed from the nasopharynx. In these forty-six cases, there was an associated

tuberculous cervical adenitis, confirmed by operation, in twenty-three instances. In the remaining twenty-three patients, no evidence of tuberculosis could be found on physical examination, nor did their history or general appearance suggest tuberculosis. During the four years from 1917 to 1921, a microscopic study of the tonsils or adenoids removed at operation in 2,260 additional cases resulted in the discovery

Fig. 9.—L. T., colored boy, aged 2, two years before operation. The mediastinal glands were enlarged, with beginning root infiltration, suggesting mediastinal tuberculosis. Physical examination was negative except for suppurative otitis media and phlyctenular conjunctivitis.

of ninety-two patients with a tuberculous infection. Of these ninety-two patients, eighteen had tuberculosis of the cervical glands, but in the remaining seventy-four cases tuberculosis was not suspected until the tonsils were studied microscopically. To summarize: The histologic examination of the tonsils and adenoids removed at operation in a series of 3,260 cases resulted in the discovery of tuberculous tonsils

or adenoids in 138 patients; eighty-five of the 3,260 patients had tuberculous glands of the neck—as proved by a microscopic examination of an excised gland—but we were able to demonstrate an associated tuberculosis of the tonsils or adenoids in only forty-six of the eighty-five cases. If we deduct the forty-six cases in which tuberculous tonsils were found in association with a tuberculous cervical adenitis from the total of 138 cases of tuberculous tonsils or adenoids, we still

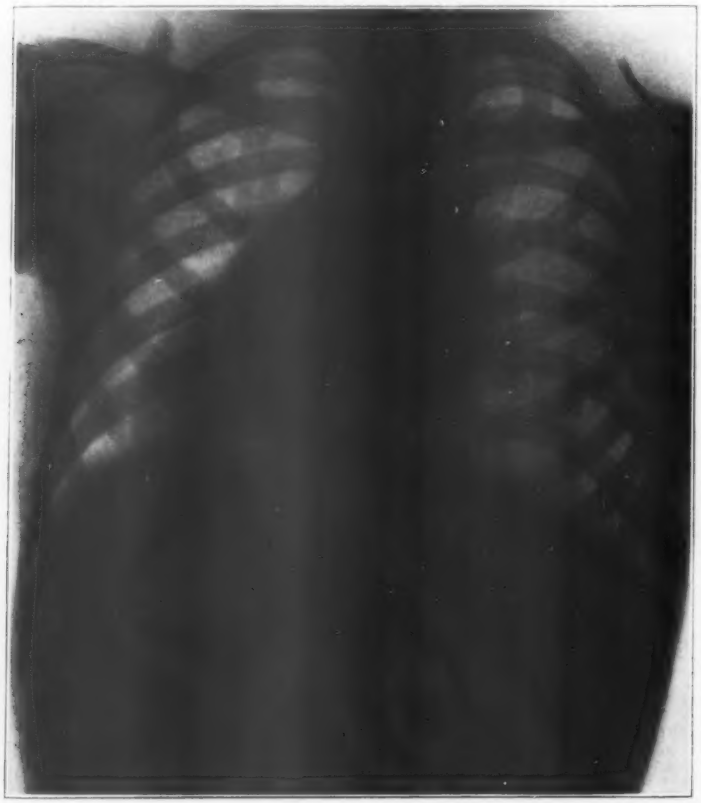

Fig. 10 (L. T.).—Five years later, the mediastinal glands were still enlarged. The infiltration had extended into the second zone of the left upper lobe. The apices above the clavicles were clear. Examination of the lungs was negative. Adenoids were found to be tuberculous; the tonsils were not tuberculous. There was no tuberculous cervical adenitis and no conjunctivitis.

have ninety-two in which there were no general or local manifestations of tuberculosis. Each of these patients applied for treatment on account of discharging ears, frequent colds or attacks of tonsillitis, and a tuberculous lesion was not suspected until the excised tonsils or adenoids were examined microscopically.

It is desirable to know the ultimate result in these patients with an apparently primary tuberculosis of the lymph-adenoid tissue in the throat. Will they subsequently develop clinical evidence of tuberculosis in the cervical or mediastinal glands, in the lungs, or elsewhere in the body? When tubercles are discovered in the tonsil by histologic examination and no clinical manifestations of the disease are evident on physical examination, should we notify the parents of the child, or the

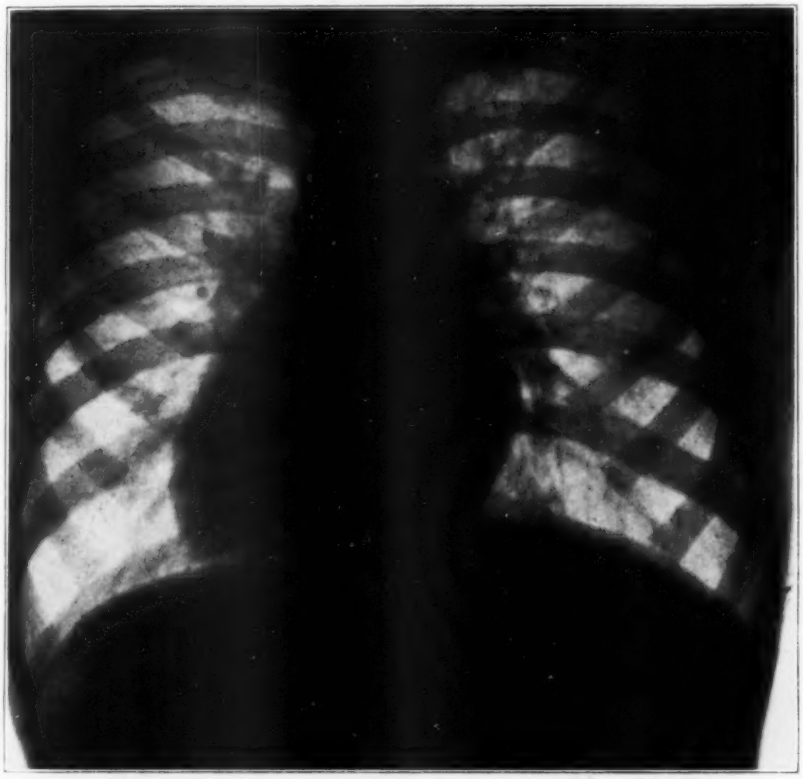

Fig. 11 (L. T.).—Two years later, the mediastinum was within normal limits. There were numerous small calcified glands in the root zone extending into the second zone. The apices above the clavicles were cloudy. The roentgen-ray impression is fibroid tuberculosis of both uppers. The lungs showed impairment of the percussion note over both upper lobes, but no changes in the breath sounds. The general health was good. The ears were normal.

patient himself if he is an adult? Is it necessary to withdraw the child from school, or send the adult to a sanatorium in order to improve the general physical condition and to educate the patient to conform to the rules for the recovery from tuberculosis? Does the discovery, through histologic examination, of tubercles in the tonsils or adenoids

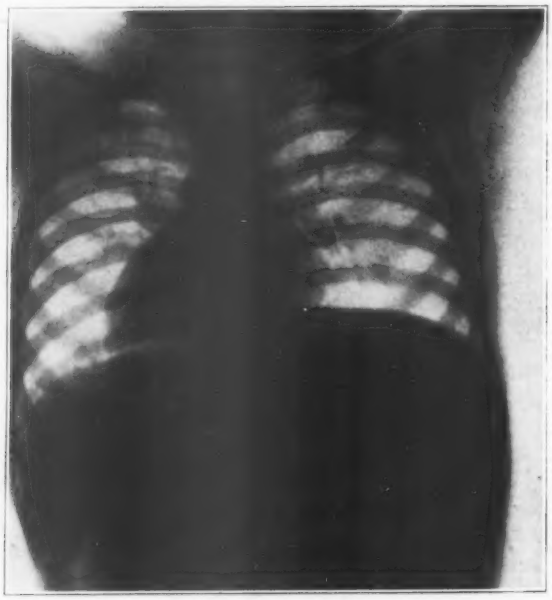

Fig. 12.—I. S., colored girl, aged 1½; before operation. The mediastinum was not enlarged. There was slight infiltration of the left upper lobe extending beyond the root zone, strongly suggestive of pulmonary tuberculosis. The lungs were clear on physical examination. There was tuberculous cervical adenitis on the left side. After the operation, the adenoids were found to be tuberculous; the tonsils were not tuberculous.

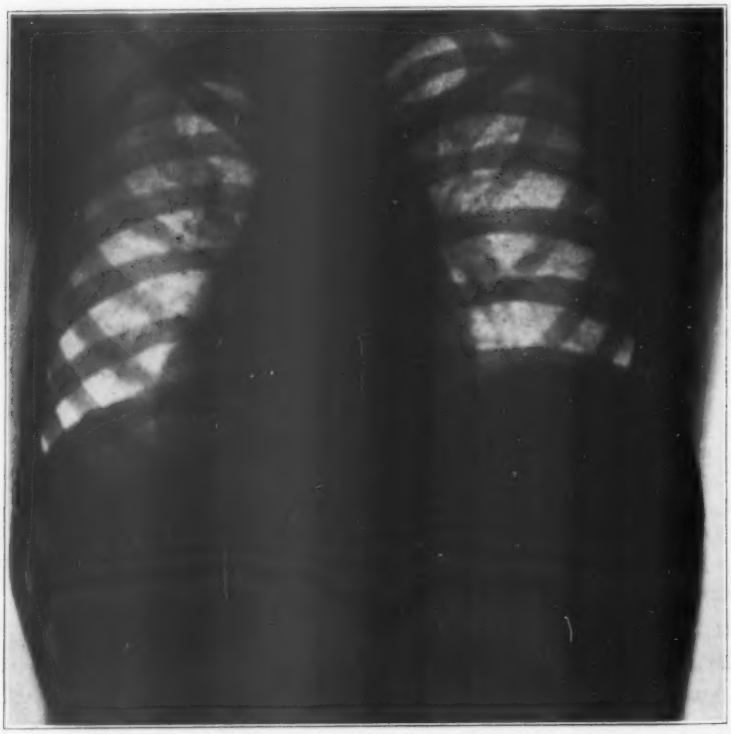

Fig. 13 (I. S.).—Five years later, the mediastinum was enlarged, with fibrosis of both upper lobes, especially the left, tuberculous in origin. A recent physical examination was negative. The cervical adenitis subsided shortly after operation. The patient appears healthy and robust.

indicate that there is already a widespread distribution of tubercle bacilli, or can we assume that the infection is local, and since it produces no local symptoms, is of no significance and may be ignored?

These are pertinent questions that any physician may be called on to answer. Our patients include physicians, nurses, students and children that have always received the best of care, as well as those from the class that visit the outpatient department of the hospital. In order to answer these questions, it will be necessary to observe a group

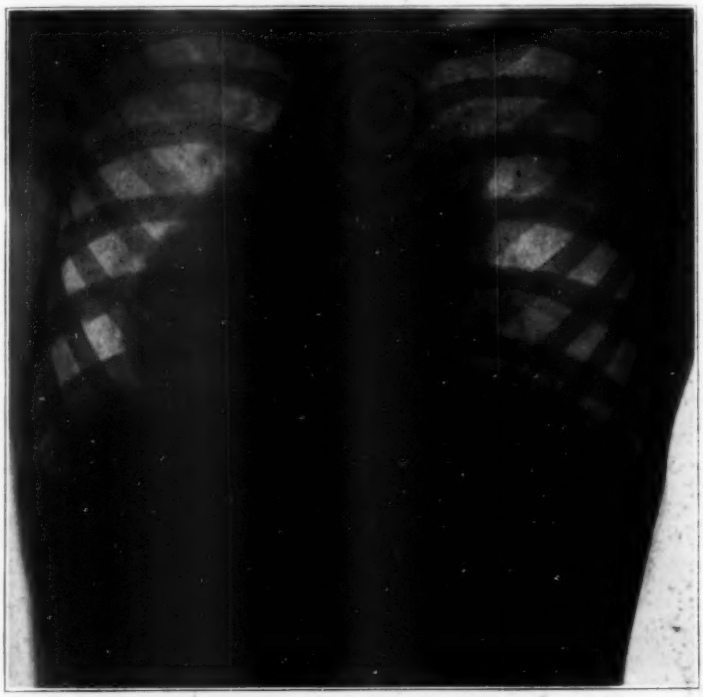

Fig. 14.— J. H., colored girl, aged 6; before operation. The mediastinal shadow was slightly enlarged. The apices were clear. The soft infiltration beneath both clavicles suggested an active tuberculosis. The patient had had no symptoms except tonsillitis. Physical examination of the lungs showed a few sonorous râles along the inner border of both scapulae, but pulmonary tuberculosis was not diagnosed. The adenoids were found to be tuberculous, but the tonsils were not.

of such patients for ten or fifteen years, with frequent physical examinations and a roentgen ray of the lungs at least once each year.

It is difficult to persuade patients that are free from clinical symptoms to return year after year for observation, but with the aid of the social service department, the Phipps department for the study of

tuberculosis, the staff of the Harriet Lane Home for children, and the special fund for the study of tuberculosis in the Johns Hopkins Hospital, we have been able to obtain accurate records, and a total of 100 roentgenograms of the lungs on fifty of our cases from the outpatient department. Each of these fifty patients had tubercles in either the tonsils or the adenoids, as shown by histologic examination; forty have

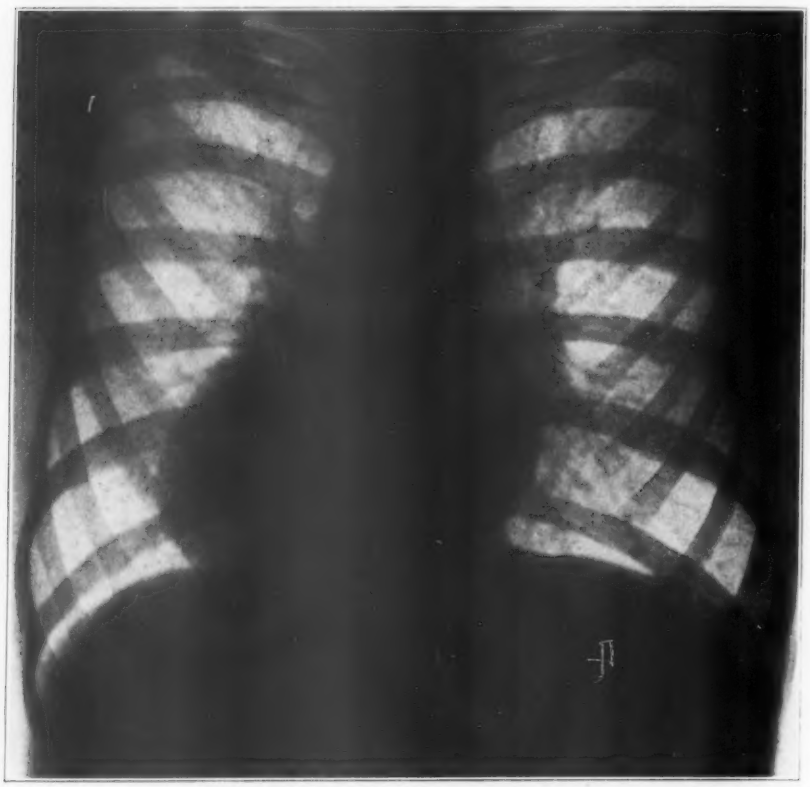

Fig. 15 (J. H.).—Five years later, both upper lobes showed decided fibrosis, suggesting end-result of tuberculosis. Repeated physical examinations during the period of observation showed no evidence of active pulmonary tuberculosis. The general health is good. The patient appears healthy and robust.

been under observation for five years or longer, and in the remaining ten cases, at least two years has elapsed since the operation.

In addition to the questions enumerated above, these fifty cases have been studied in order to determine the following points:

1. Is there any roentgen-ray evidence of tuberculosis in the lungs or the mediastinal glands? And if suspicious lesions are found, do they progress or retrogress during a five-year period of observation?

2. Has the use of ether anesthesia been detrimental?

3. Do the children that had a positive tuberculin test at the time of their first examination, prior to operation, and in whom tuberculous tonsils or adenoids were found when the excised tissues were examined histologically, have a negative reaction at any time subsequent to the operation?

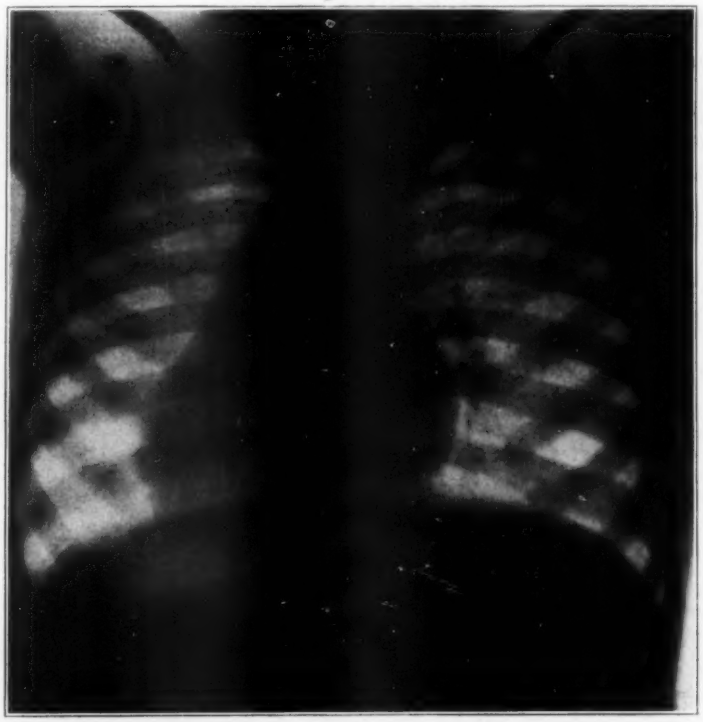

Fig. 16.—R. B., colored boy, aged 5. One year after operation. The mediastinal glands were enlarged, suggesting mediastinal tuberculosis. Physical examinations at the time the roentgenograms were made and before operation were negative except for a tuberculous cervical adenitis on the right side. Only the right tonsil was found to be tuberculous.

SUMMARY OF POSTOPERATIVE OBSERVATIONS

Following is a brief summary of the information we have acquired from the postoperative observation of the fifty cases mentioned above:

Tuberculosis of the Cervical Lymph Glands.—This condition developed in only two patients during the postoperative period of observation. One was a white woman, aged 27, who first came to the outpatient department in 1914, complaining of frequent attacks of tonsillitis. Tuberculosis was not suspected until the microscopic sections

of the tonsils were studied. This patient has had numerous physical examinations and roentgen-ray examinations of the chest during the nine years since her tonsillectomy, but no evidence of tuberculosis was discovered until the glands at the angle of the jaw on the right began to enlarge, eighteen months after the operation. The tuberculous glands were removed, and she is now apparently perfectly well. No clinical or roentgen-ray evidence of tuberculosis of the lungs has ever been discovered.

Fig. 17 (R. B.).—Three and one half years after operation, the lungs were clear. The mediastinal glands were large and tuberculous. The cervical glands on the right were excised two years ago. No further symptoms developed. The general health is good and a recent physical examination was negative.

The other patient that developed tuberculous cervical adenitis during the postoperative period of observation was a white child, aged 3 years, who was brought to the outpatient department, in 1919, with phlyctenular conjunctivitis. The father had died of tuberculosis in 1918. The tonsils and adenoids were removed, and on microscopic examination, tubercles were found in each of the tonsils and in the adenoid tissue. Tuberculous glands appeared on the left side of the neck three

months after operation; they were incised and curetted, and have gradually disappeared. At present, over four years after the tonsillectomy, this child is apparently in perfect health.

Tuberculosis of the Mediastinal Lymph Glands.—This condition has been diagnosed in our series from the roentgenogram of the chest. In three children, the roentgenogram made before operation showed a normal mediastinum. The tonsils were removed, and were found to be tuberculous, and subsequent roentgenograms showed enlarged medias-

Fig. 18.—G. C., colored girl, aged 4. One year before operation. The enlargement of the mediastinal glands with fibrosis at the left apex were tuberculous in origin. Two years before operation, the child developed scattered tuberculids following an incision of a tuberculous gland in the right side of the neck. The tuberculids disappeared before operation. Only the right tonsil was tuberculous. Repeated physical examinations of the lungs were entirely negative.

tinal glands. Each of these children was under the observation of the pediatric department, and the home conditions were improved by visiting nurses. In a colored child, first seen at the age of 1½ years, the enlarged mediastinal glands were first noticed 5 years after the discovery of the tuberculous infection in the tonsils. In a white child, first seen at the age of 3 years, the enlarged mediastinal glands were

first noticed three years after operation. The remaining patient, a white child, aged 6 years, had no roentgen-ray or clinical evidence of a mediastinal lesion before operation, but a roentgenogram taken six months later showed definitely enlarged mediastinal glands. A roentgenogram made five years after operation showed that the enlarged glands had subsided and the mediastinum was again normal. These three children have grown and developed normally, go to a public school and are apparently perfectly well.

Tuberculosis of the Lungs.—During the postoperative period of observation, pulmonary tuberculosis developed in five patients—three children and two adults. In only one, a child, aged 7 years, is there any clinical symptom of the lesion in the lung that was diagnosed from the roentgenogram as pulmonary tuberculosis. In the remaining four patients, the roentgen-ray changes in the lungs that were first noted two years after operation gradually became fibroid in character.

No one of the four other patients mentioned above ever had fever, cough, loss of weight, anorexia or any other of the common symptoms of pulmonary tuberculosis. The one child that had both roentgen-ray and clinical evidence of a pulmonary lesion is apparently cured. For several years, she was an inmate of an open-air school, but she has now returned to the city and goes to a public school.

Tuberculosis of the Bones and Joints.—This condition appeared in two patients, one an adult and one a child. In each case, tuberculous tonsils or adenoids had been removed from one and one half to four and one half years before the onset of the joint symptoms. A white man, aged 22 years, had his tonsils and adenoids removed in 1917, on account of a tuberculous cervical adenitis. Microscopic sections showed tubercles in both of the tonsils, in the adenoids and in the excised glands. A psoas abscess and a tuberculous lesion in the third lumbar vertebra appeared one and one half years after tonsillectomy. Drainage of the abscess and an Albee graft by Dr. W. S. Baer have resulted in an apparent cure. In a white child, aged 2 years, the adenoids alone were removed on account of a chronic suppurative otitis media. The ear condition rapidly healed. Symptoms of tuberculosis of the hip first appeared four and one half years after the adenoidectomy. The child was greatly improved by fixation and traction of the affected leg, and there is no clinical evidence of tuberculosis elsewhere in the body.

Cervical Adenitis Group.—A roentgen-ray diagnosis of tuberculosis of the mediastinum or lungs was made in thirteen out of a group of eighteen patients in whom the tonsils and adenoids were removed because of a definite tuberculous cervical adenitis. In the remaining five patients, repeated physical and roentgen-ray examinations, over a period of five years, have revealed no involvement of either the medi-

Fig. 19 (G. C.).—Four years later, the large mediastinal glands with fibrosis at the left apex were still noted. The cervical glands were still visibly enlarged, but they have caused no symptoms for past two years. The patient's health is good. A recent physical examination of the lungs was negative.

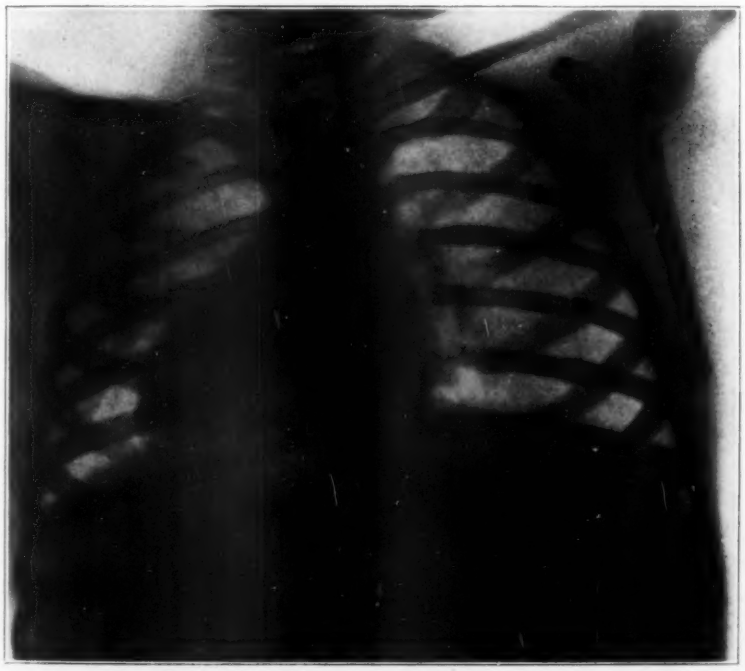

Fig. 20.—N. N., colored boy, aged 6, two years after operation. The mediastinal glands were slightly enlarged, suggesting a mediastinal tuberculosis. The lungs were clear except for occasional scattered râles. The patient had been under constant observation since 1 year of age, for chronic bronchitis. After operation, both tonsils were found to be tuberculous.

astinum or the lungs. In five of the thirteen patients having cervical adenitis, with mediastinal or lung involvement, a series of roentgenograms show a progression of the lesion during the five year period following the removal of the tuberculous tonsils. Only two of these five patients developed clinical symptoms. One is dead, and the other is now free from symptoms. In three of the thirteen cases mentioned above, tuberculous lesions appeared in the roentgenograms made during the period of postoperative observation. In one, enlarged mediastinal glands, with no evidence of a pulmonary lesion, was first noted three years after tonsillectomy. In one, the roentgenogram made before operation revealed a slight fibrosis in the lung, but enlargement of the mediastinal glands was first noted five years later. There have been no clinical symptoms referable to the lungs in either of these patients. In all of the patients mentioned in this paragraph, the indication for tonsillectomy was an evident tuberculous cervical adenitis, and in each of these eighteen cases, the tonsils were proved tuberculous by histologic examination.

Summary of Roentgen-Ray Examinations.—Our data in regard to the involvement of the mediastinum or lungs in the group in which no tuberculosis was suspected at the time of the tonsillectomy is based on a clinical and roentgen-ray study of twenty-two cases. In ten patients, no roentgen-ray or clinical evidence of a mediastinal or lung lesion has been found during the five years of postoperative observation. In six patients, the enlargement of the mediastinal glands or the roentgen-ray evidence of a lung lesion, shown in roentgenograms taken soon after the tonsillectomy, has entirely disappeared; while in the remaining six patients, the lesions in the chest have progressed according to the roentgenograms, but no one of these six has had fever, cough, râles or other evidence of a clinical tuberculosis.

Summary of Incidence in the Johns Hopkins Hospital.—The general incidence of tuberculosis of the tonsils and adenoids as noted in the literature approximates 5 per cent.; while in patients with manifest tuberculous cervical adenitis, the incidence varies from 20 to 72 per cent., depending on the technic. In this series, the general incidence is 4.18 per cent., while it rose to 48.2 per cent. in the patients with tuberculous cervical adenitis. Among patients with no clinical evidence of tuberculosis, it falls below 2 per cent. Although not shown in Table 2, tuberculosis of the tonsils and adenoids occurred four times as frequently in the colored as in the white race.

It can be seen from Table 3 that more than half of the tuberculous patients were under 10 years of age. These figures stand in marked contrast to those of Weller, who found the greatest incidence in young adults. Might not this increase in the incidence among very young

children tend to connect tuberculosis of the tonsils and adenoids with the primary tuberculous infection of childhood rather than with the clinical tuberculosis of young adults?

The use of ether anesthesia for the removal of tonsils and adenoids has been most satisfactory in our experience, and we see no reason why we should substitute gas and oxygen or a local anesthetic. With the exception of a few cases in which a cardiac, renal or pulmonary condition contraindicated the use of a general anesthetic, we have employed ether in all of the 3,260 cases mentioned in the early part of this paper. We do not know of a single case of postoperative lung abscess in this

TABLE 2.—*General Incidence of Tuberculosis of Tonsils and Adenoids*

Symptoms	Operation	Number of Patients	Tuberculous Lesions		Percentage
No clinical tuberculosis.....	Tonsillectomy and adenoidectomy	2,215	Tonsils or adenoids	72	3.25
No clinical tuberculosis.....	Adenoidectomy	1,000	Adenoids	25	2.5
Tuberculous cervical adenitis....	Tonsillectomy and adenoidectomy	85	Tonsils or adenoids	41	48.2
Total.....		3,300		138	4.18

TABLE 3.—*Age Incidence*

Years	Tonsillectomy and Adenoidectomy		Adenoidectomy	
	Number	Percentage	Number	Percentage
1 to 10.....	60	6.52	19	3.92
10 to 20.....	32	4.7	5	2.3
Over 20.....	21	3.0	1	1.1
Total.....	113	4.91	25	2.5

series of 3,260 patients; and the hospital records show only three cases of postoperative pneumonia, with recovery in each instance within ten days. Finally, the records that are summarized above of the fifty patients with a tuberculous infection and the post-operative condition of the remaining eighty-eight patients with tuberculous tonsils or adenoids that are not included in this paper, indicate that a latent tuberculous infection of the tonsils, adenoids or cervical or mediastinal glands is no contraindication to the use of ether anesthesia. We wish to emphasize the fact, however, that the anesthetic is always administered by a professional anesthetist. After the mouth gag is introduced, ether is never given in an amount sufficient to abolish the swallowing reflex, and the greatest care is taken to prevent the aspiration of blood, vomitus or bits of tissue.

We are indebted to Dr. F. H. Baetjer, who interpreted the roentgenograms. The entire group of 100, some dating back ten years, was assembled in May, 1923, and Dr. Baetjer dictated his impression without any knowledge of the clinical condition of the patient. We are also indebted to Dr. R. J. Dunn of Sumter, S. C., who assisted in assembling the records and microscopic sections.

Fig. 21 (N. N.).—Three years later, the lungs were clear. The mediastinal glands at the root of the right hilum were calcified, indicating a healed process. The general health has been good. The patient showed no signs of pulmonary tuberculosis during the period of observation.

If the roentgen-ray changes in the lungs and mediastinal glands are sufficient to establish a diagnosis of tuberculous infection when the patient himself is free from all signs and symptoms of such an infection, then three-fifths of the fifty patients studied in this series had tuberculosis elsewhere in the body prior to the discovery of the lesion in the tonsils or adenoids. Our impression, based on the postoperative clinical study of this group for five years or longer, is that a tubercu-

lous infection of the tonsils and adenoids is of but little significance and might be best ignored. It is true that a continued observation of this group for another five or ten years might result in finding an abnormally high percentage of clinical tuberculosis, but our results up to the present lead us to doubt the wisdom of notifying the patient or the family that the tonsils or adenoids, on histologic examination, show

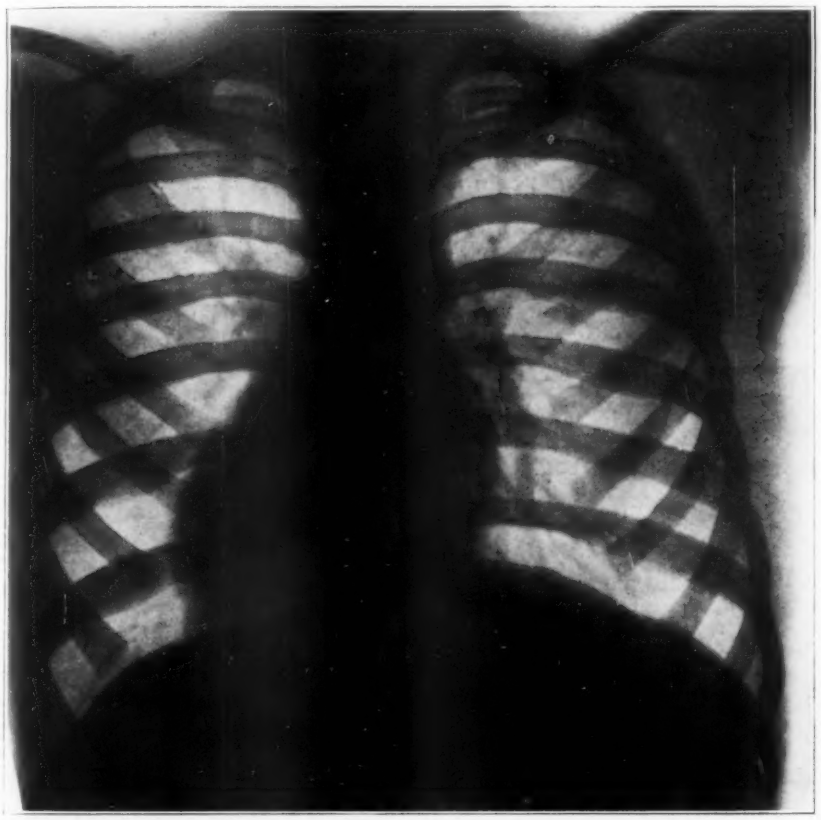

Fig. 22 (N. N.).—Eight years after operation, the mediastinum was normal. There was absorption of the root shadows. The calcified glands at the root of the right hilum were still present. The patient was in the hospital with typhoid fever. Nothing to suggest tuberculosis was found in the examination. At present the patient appears healthy and robust.

tubercles, and thus stigmatize the patient as tuberculous. In the past, we have always made such notifications, and in many instances have thus caused an unnecessary loss of time from work or study; an expenditure of funds that could be ill-spared; a large amount of mental worry to the patient, family and friends, and finally; a blot on the record if life insurance is applied for at some later date.

CONCLUSIONS

We have succeeded in obtaining a series of postoperative observations on fifty patients from the outpatient department of the Johns Hopkins Hospital. Forty have been observed from five to ten years, and ten for at least two years. Each of these patients has had a roentgen-ray examination of the chest and a general physical examination at least once a year since removal of the tonsils and adenoids. In each of these patients, the tuberculous lesion in the tonsil or adenoid tissue was verified by the pathologic department.

It is our impression that:

1. Tuberculosis of the tonsil is usually a bovine bacillus infection. We base this conclusion on the age incidence (over 50 per cent. of our patients were less than 10 years of age); the frequency of the involvement of the cervical and mediastinal lymph glands; the strikingly low incidence of pulmonary, intestinal or other complications during the five to ten year period of postoperative observation, and the growth, appearance and excellent general physical condition of these children in spite of the roentgen-ray evidence of tuberculosis of the mediastinum. It is possible that such a tuberculous infection of the lymph-adenoid tissue in the throat, if not complicated with frequent secondary infection, is of value from the point of view of immunity.

2. When tubercles are found in the tonsil on histologic examination, it is needless to become unduly excited, because the infection is already widespread in the cervical, mediastinal and probably the mesenteric lymph glands.

3. In such cases, the removal of tonsils and adenoids is of value in that it reduces the secondary infection, but there is no evidence that by operation on the throat one can remove the only tuberculous focus in the body.

4. Tuberculosis of the tonsils or adenoids is never recognizable from the gross appearance before operation unless there are superficial ulcerations secondary to an open pulmonary lesion; and when the lesion is discovered microscopically, it is probably not advisable to alarm the family, stigmatize the patient as tuberculous or seriously interfere with his ordinary routine duties, since the majority of such patients will probably never have clinical symptoms of the disease.[20]

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20. Because of lack of space, a number of references in the form of a bibliography have been omitted. These are included in the reprints, a copy of which can be had on application to the author.

BLINDNESS AND OTHER DISEASES IN CHILDREN ARISING FROM DEFICIENT NUTRITION (LACK OF FAT-SOLUBLE A FACTOR)*

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While blindness in children has been on the decrease in the greater part of the civilized world, the opposite is true in Denmark. Blindness in children has increased not only absolutely but also relatively to the population. Many causes have contributed to the diminution of blindness in other countries, but the chief one undoubtedly is that gonorrheal ophthalmoblenorrhea in the new-born has become a rare disease as a result of the special measures which have been adopted against it everywhere. That gonorrheal ophthalmia has become a rarity is also true in Denmark, but in spite of this the percentage of blindness in children is increasing.

There is, however, another disease of the eye peculiar to childhood which leads to blindness: xerophthalmia. It was first described in the middle of the last century by German ophthalmologists. According to all previous reports, xerophthalmia had a serious prognosis, as it occurred particularly in the final stage of a cachectic condition due to a protracted wasting disease such as tuberculosis, syphilis, typhoid fever and acute or chronic digestive disease, and especially in badly nourished and debilitated children.

In the middle and at the close of the last century and in the beginning of this, "epidemics" of an eye disease were described which were undoubtedly identical with xerophthalmia. These epidemics occurred among negro children in Brazil, during periods of fasting in Russia and when famine and summer diarrhea appeared together in Japan, and it was always the badly nourished and diseased children of the poorer classes who were attacked. The majority of the authors who have described these epidemics therefore associate xerophthalmia in children with a poorly selected and deficient diet. Primitive medicine in Japan, Russia and other countries has from ancient times treated this eye disease with fat substances, especially cod liver oil and liver, and Mori has declared that the disease is due to lack of fat in the diet.

In Europe, xerophthalmia is considered extremely rare. A few cases have been recorded in badly nourished children in England and Denmark, while in Germany Czerny and Keller, and, according to them,

* Received for publication, Nov. 21, 1923.

several others, have now and again observed xerophthalmia in the final stage of the nutritional disease they call "Mehlnährschaden."

The children's department of the Danish State Hospital was opened in 1911. By the next spring a number of children had been admitted with a curious eye disease which proved to be xerophthalmia, and in the following year the number increased to an alarming extent. I thus had the opportunity of studying this disease in its different forms and stages, about which I published communications in 1914, 1916, 1917, 1918 and 1922.

My investigations indicated that the disease was due to a lack of a particular substance. This substance was present in fresh milk, but not in separated milk or in buttermilk. It was therefore united with the butter fat, and it was also found in abundant quantities in cod liver oil. My investigations further showed that the disease could not be attributed to the absence of fat as such, because even though the children had received margarin or pork fat, they could easily contract xerophthalmia.

In England and the United States, a large number of experimental investigations on nutrition were carried out, partly before and partly at the same time as my work. From these inquiries, we became further acquainted with the cause of xerophthalmia and the disease associated with it. It was from the animal experiments of Osborne and Mendel, and especially of McCollum and his collaborators, that we now know the cause of xerophthalmia to be the lack of the fat-soluble A vitamin. This factor, which is united with certain fat substances and is notably found in large quantities in butter and cod liver oil, must be constantly supplied to the organism, particularly the growing organism, in order that growth shall take place. Deprivation for a long period leads to xerophthalmia and death.

When a child, particularly one in the first few years of life, is deprived of its mother's milk or fresh milk, as well as cream and butter, and is kept for a long time on a diet consisting of puddings made with separated or buttermilk, bread and other farinaceous foods, mashed potato, a little pork and other meat, margarin and pork fat, it will become ill as a result of the absence of the fat-soluble A factor. Even the consumption of a little vegetable, fruit, and green stuff now and then, will not help much, for a small child can digest only trifling amounts of green stuff.

A deficiency of the fat-soluble A factor may also occur in children who have received a quantity of pure milk that is stale and modified adversely, the fat-soluble A factor being destroyed by oxidation and repeated heating.

The disease is ushered in by failure to gain weight. Later on, there is a loss of weight and the child is quiet and apathetic. In other words, there is inhibition of growth and depressed psychic and bodily vitality.

These children are liable to be attacked by some infectious disease, which usually takes the form of a simple catarrhal infection of the mucous membranes, particularly of the respiratory organs and middle ear. *B. coli* infections of the urinary tract and infectious dermatitis

Fig. 1.—Child with xerophthalmia.

are also frequently observed. These infections are extraordinarily persistent and often have a fatal issue. An increased susceptibility and a diminished power of resistance to infections are therefore present.

The eye lesion does not appear as a rule until late in the disease. The first symptom is night-blindness (hemeralopia) and dryness of the ocular conjunctiva (xerosis). The dryness later extends over the cornea, which becomes hazy, shrivels up, and shortly after, pieces of it

become necrosed (keratomalacia). In most cases, the ocular conjunctiva is infected; the child then has photophobia; the conjunctiva is red and swollen, and the eyes begin to water. When the cornea necroses, the inflammation spreads to the interior of the eye. The inflammation may be so intense and widespread in many cases that it almost obscures the original xerosis. The eye lesion can with difficulty be distinguished from the destructive inflammation in such cases.

If the disease is not recognized in time, the child becomes blind. If the child does not then die of a secondary infection, the most frequent cause of death is bronchopneumonia.

Fig. 2.—Child with xerophthalmia, showing eye lesion.

A boy (Fig. 1), aged about 2 years, appeared to be a healthy child, except that the eyes were swollen and kept tightly closed. He was small and under weight, and had a catarrhal infection. His eyes were completely destroyed by the disease, as is shown in Fig. 2. The conjunctiva was swollen and red, both corneas were necrotic and there was pus in the anterior chamber. The child was cured, but became totally blind, as the cornea never heals with clear transparent tissue.

The scars are always hazy and opaque, as will be observed from the succeeding pictures of children who were cured (Figs. 3 and 4). In cases in which the cornea is only partly damaged, the child can usually be saved from complete blindness.

Fig. 3.—Recovery from xerophthalmia, showing permanent eye location.

Fig. 4.—Recovery from xerophthalmia, showing permanent eye lesion.

If a small child receives no milk whatever, not even separated milk or buttermilk, but is given only water, tea, gruel and the like for a prolonged period, on account of diarrhea or some other malady, it will become wasted and atrophic, with dry wrinkled skin.

In this case, in addition to the absence of the fat-soluble A factor, several others were missing, but it is essentially the primary wasting disease, in this instance the intestinal trouble, which dominates the entire clinical picture. Such a case resembles most nearly the xerophthalmia described by the older authors.

When the child is a little older and stronger, and not ill, it can apparently thrive for a long time on infant food or other essentially carbohydrate diet, but sooner or later the tissues become edematous, and if the child has received no fresh milk, xerophthalmia, bronchopneumonia, etc., may supervene, just as in the first type.

Fig. 5.—Atrophic child.

If the child has had only a little butter in addition to the carbohydrate diet, edema, such as that in chronic interstitial nephritis, can certainly occur, but neither xerophthalmia nor marked lessening of the powers of resistance is observed, and the child is easily restored to health when it receives milk. Figure 6 shows such a case, with an enormous amount of edema but with no eye lesion.

Besides the xerophthalmia, the characteristic symptoms in this disease, which arises in consequence of the absence of the fat-soluble A factor, are, therefore, inhibition of growth and diminished resistance and vitality. In older children and adults, who hardly utilize so much fat-soluble A factor as the small child, the disease may be attended merely by the latter symptoms. It is only when the lack of the fat-soluble A factor in the food has been almost absolute for a long time, or when the bodily requirement of it has been very great, e. g., in chronic infectious diseases, that older children develop xerophthalmia. It is chiefly young children, that is to say those under 2 or 3 years of

age, that develop xerophthalmia, and this may happen even though there has been some fat-soluble A factor in their food.

During the war, there was much sickness among the older children in central Europe, which took the form especially of inhibition of growth, and a large number of infectious diseases running a grave course. Thus, many children were attacked by tuberculosis. The smaller children, on the other hand, were little affected, and xerophthalmia was scarcely ever seen in them. This state of affairs was due to the fact that during the war all the milk and milk products in Germany were rationed so that every little child obtained a certain amount daily. If the child was being suckled by the mother, she got the milk, and xerophthalmia was never observed in children suckled by a mother capable of yielding sufficient milk.

Fig. 6.—Edematous child.

In Denmark the conditions, as mentioned, were different. Before, and especially during, the war, there were many cases of xerophthalmia. In a recent investigation undertaken for the Danish Ophthalmologic Society by Olaf Blegvad, from 600 to 700 cases of xerophthalmia were found to have occurred between 1909 and 1920 among children, in a population of less than 3,000,000, and there were probably others.

The reason xerophthalmia was so widespread, despite the fact that Denmark did not take part in the war, is that this dairy-farming country manufactures and exports butter. During the war, butter, and consequently fresh milk, became so expensive that the poor could procure scarcely any of it, and they were able to obtain only separated and buttermilk, which could always be had at a low price. The number of cases of xerophthalmia increased, therefore, until 1918. In that year,

the disease was suddenly checked, as will be seen from Figure 7. The reasons were several, but the principal cause was undoubtedly that in 1918, on account of the German blockade, butter was rationed in Denmark, so that every individual, including small children, was entitled to 0.25 kg. of butter a week, and as there was practically no pork fat or margarin in the country everybody had to eat butter. Xerophthalmia ceased simultaneously. These facts show as clearly as an experiment that the absence of the fat-soluble A factor is the cause of xerophthalmia.

After the termination of the war, the rationing of butter came to an end, and xerophthalmia returned, but its occurrence was rare as compared with its former incidence. It was particularly noticeable that the

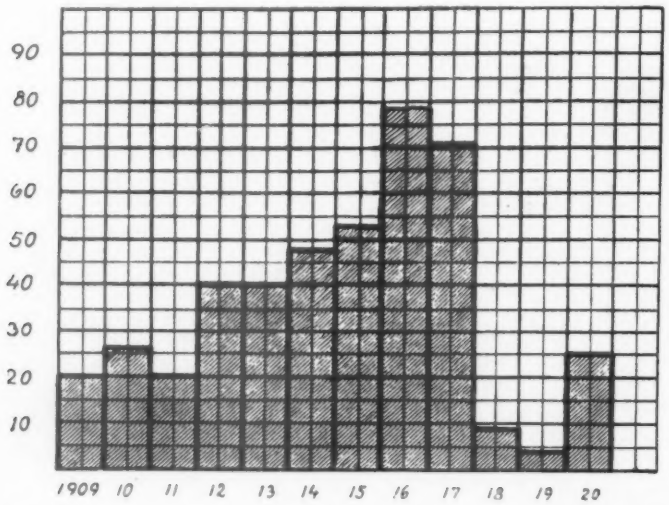

Fig. 7.—Frequency of xerophthalmia in Denmark (1909-1920).

severe cases no longer occurred. The people and the Danish physicians had come to know the disease and had learned how to avoid and treat it.

The condition caused by lack of the fat-soluble A factor is cured by supplying the latter to the patients, best in the form of fresh milk, cream, butter or cod liver oil. The xerophthalmia is the first symptom to disappear. Then the child begins to thrive and infections are gradually overcome. The latter are therefore cured by the administration of the fat-soluble A factor. This is in harmony with the old clinical experience that chronic infectious diseases, particularly tuberculosis, are improved by a diet rich in fresh milk, butter and cod liver oil.

The fat-soluble A factor is thus particularly important not only for the eye lesion but also for establishing immunity, and it is clear that a great deal of it is required in infectious diseases.

Large quantities must also be consumed during the period of growth. This is most clearly evident from McCollum's animal experiments, but the study of xerophthalmia points in the same direction, as it occurs practically only in children; that is to say, in growing individuals. Moreover, the disease appears especially in the spring months, at which season the most marked growth of the child takes place, according to Danish investigations. The cases not occurring in this annual period of growth have always been complicated by serious infections or chronic diarrhea. This distribution of the cases is shown in Figure 8.

It has been thought that the increase in the number of cases in the spring was due to a different content of fat-soluble A factor in the

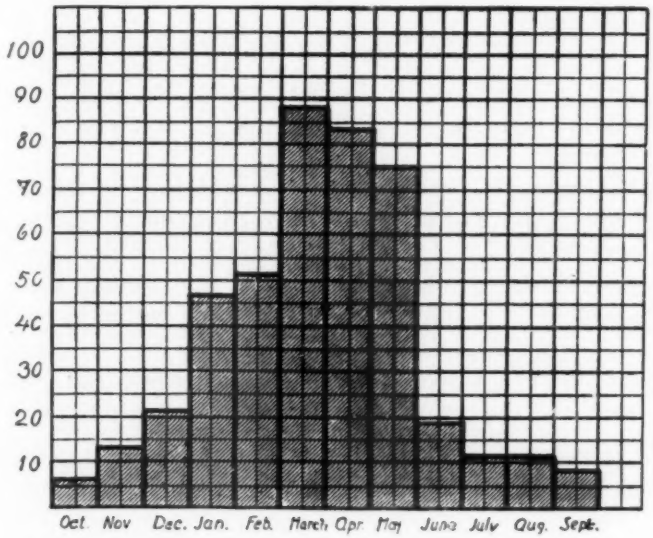

Fig. 8.—Seasonal variations of xerophthalmia in Denmark.

winter milk (stall feeding) and the summer milk (grass feeding). It is possible that there is more of the fat-soluble A factor in the first "grass milk," and that this has something to do with the disappearance of the disease in the summer, but a decreased fat-soluble A content in the milk cannot be the cause of the many cases arising in the growth-period of the spring because precisely the same distribution of cases is found among patients that have received no milk or milk products at all for a long while. It is also difficult to believe that milk from healthy cows could be deficient in the fat-soluble A factor. At any rate, it has not yet been proved, and the experience of xerophthalmia in Denmark is against the supposition.

This special relation between the fat-soluble A factor and growth, and between it and infections, has not been thought to be confined to

this body, but is also found in other deficiency diseases. Thus, Alfred F. Hess has especially pointed out that the relation is somewhat similar in the case of scurvy, the disease which arises from the absence of the antiscorbutic factor. Scurvy, like every other serious disease, is undoubtedly able to affect growth and diminish the power of resistance against infections; but in the disease caused by the lack of the fat-soluble A factor, both these features are very strongly emphasized, and in a special manner. Thus, scurvy, in contradistinction to xerophthalmia, is not particularly associated with childhood, nor does it, like xerophthalmia, occur especially during the annual growth period. Moreover, a statistical examination of my cases of scurvy shows that this disease is not associated with infections to nearly the same extent as is xerophthalmia.

CONCLUSIONS

Xerophthalmia and rickets are very rarely met with in the same child. Rickets can therefore hardly be due to the absence of the fat-soluble A factor.

What I have said here and have proved to you testifies to the enormous importance of milk as a food for the child. No other article of diet can replace milk. Absence of milk from the diet or the inclusion of improperly modified milk is the origin of most serious diseases.

By employing milk, and especially cream and butter, in the diet, not only is this terrible eye disease, which I believe will be discovered in every country when it is looked for, cured, but growth and development are promoted, and the cure of our most prevalent infectious diseases is advanced.

A CLINICAL STUDY OF RICKETS IN THE BREAST-FED INFANT *

L. R. DEBUYS, B.S., M.D.

NEW ORLEANS

In the practice of pediatrics, one frequently finds rickets in the breast-fed infant. On close questioning, there may or may not be elicited some error in the feeding. If there is an error, it is likely to be blamed for the disease; and if there is no error, the history is very likely to be regarded as doubtful as regards some points. This study was undertaken to ascertain the incidence of rickets in breast-fed babies, and to investigate the symptoms and the influence of the disease in those whose feeding and care was supervised and directed from birth.

In my service of the newly born, with its followup clinic, at the Touro Infirmary, an opportunity is afforded to study those infants whose care is begun at birth and closely supervised and regulated thereafter. This service has been described elsewhere.[1] Suffice it to say that it includes an inside and an outside division and a followup outpatient department. The inside division includes (*a*) the care of all the infants born in the obstetric service, (i. e., the free patients), with the responsibility for the baby's welfare beginning at the time the cord is cut; and (*b*) the complete supervision of the breasts of the mother. The outside division in like manner includes those babies born in the home and delivered by the outside obstetric service of the institution. These babies report to the outpatient followup clinic on the Thursday nearest which they are 4 weeks old, and return every fourth Thursday thereafter. There is a social service operating in connection with the department so that a close association with the families is had.

Those eligible to this free service include families whose earnings do not exceed $100 a month.

The inside division includes only white patients, there being no provision for colored patients; the outside division, both the white and colored. The service, then, includes members of both races whose earning capacity it is believed warrants free care.

This service of the newly born, which was begun nearly two years ago, is increasing rapidly in size. This study extended over the period of the first year of its operation. Recently, our maximum attendance was reached when, on one Thursday morning, ninety-one babies were seen.

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* From the service of the newly born, Touro Infirmary, and the Department of Pediatrics of the School of Medicine of Tulane University of Louisiana.

1. DeBuys, L. R.: South. M. J. **15**:115-120 (Feb.) 1922.

In the observations included in this study, routine examinations were made of each and every baby for clinical evidences of rickets. These observations were made by another observer and checked by me, the readings being practically always the same.

Rickets is an extremely interesting disease and one of which it may be said that the more it is studied, the more obscure its etiology appears to be. It has its pathologic, serologic, roentgenologic and clinical findings. Routine observations in this study included rickets only in its clinical manifestations. The clinical evidences of rickets included were bosses, craniotabes, costal beading, lateral flattening of thorax, flaring ribs, epiphysial enlargements and bow-legs. Observations were also made on the growth and development of the children investigated, including measurements of height, weight, head, chest and abdomen, and their period of dentition. The ages at which they sat alone, stood alone and walked alone were also noted, as well as their condition of general nutrition.

This study included observations covering a period of one year, made on 197 babies, sixty-two white and 135 colored. Twelve calendar months were selected as the period, in order to include the seasonal influences.

It had been found that greater cooperation and better results were obtained with the method of feeding employed as a routine in the pediatric service of the institution; hence, it was chosen as a uniform one in this investigation. It consisted in a two hour interval feeding for those babies whose weights were under 9 pounds (4,082.3 gm.); a two and one-half hour interval for those whose weights were between 9 and 12 pounds (5,443 gm.), and three hour interval for those weighing 12 pounds and upward, only one breast being fed at a time and no feeding exceeding twenty minutes. This did not mean that each baby had to be kept at the breast for the twenty minutes, but only so long as he showed an inclination to nurse vigorously. Occasionally a baby is encountered who has to be encouraged to nurse, and in these instances, the length of the feeding was not to exceed twenty minutes. We have shown to our satisfaction that, in the twenty-minute nursings, the bulk of the food is usually obtained within the first ten minutes, and that in the second ten minutes very little food was taken. The last milk obtained, however, consists of strippings, which are rich in fats, and this element being necessary, the twenty-minute feedings were advocated. When the breast milk was thought to be insufficient because of no gain in the baby's weight, and there was no disease in the patient, observations were made on the breasts by weighing before and after nursing. When necessary, complemental feedings were added. Seventy-four per cent. of the white mothers were able to nurse their babies exclusively for three months; 37 per cent. were able to nurse

their babies exclusively for six months. Seventy-eight per cent. of the colored mothers were able to nurse their babies exclusively for three months; and 20 per cent. were able to nurse their babies exclusively for six months.

As a rule, cereal jellies and toast were begun when the baby was 6 months old, or when its weight was 15 pounds (6,803.9 gm.). Orange juice was invariably begun as soon as any food other than breast milk was added to the baby's diet. Broths were begun at about 9 months, and strained vegetables toward the end of the year.

In no case was cod liver oil given before marked evidence of rickets was noted. In only thirty-nine instances was it begun within the first three months, twelve of which were in white and twenty-seven in colored children; in seventy-five instances, before the sixth month, twenty-six of the babies being white and forty-nine colored, and in 107 instances in the series, cod liver oil was begun before the twelfth month, thirty-nine of the babies being white and sixty-eight colored.

The diet of the mother was not restricted. Fruits and vegetables in season, as well as beans and peas and meats, constituted the major part of the dietary, this being, of course, influenced by the income.

Hygienic measures other than those having to do with the feeding, and including airing, bathing, exercise, clothing, rest and habits were advised, and in many instances, the cases were referred to the social service for investigation and supervision.

It was noted that those babies who remained away from the outpatient followup clinic returned invariably with evidence of more advanced rickets than was found in those cases constantly under supervision for a like period. It was also noted that in these cases there was a direct relation between the severity of the rickets and the interval of nonsupervision. In only two instances were the babies referred to the general clinic because of not following directions. Both children were colored. Only forty-two of the babies suffered some minor acute illness during the entire year. Thirteen of these were white and twenty-nine colored. There was no relation between these acute ailments and any increase in the symptoms of rickets.

Of the white babies studied, 47 per cent. were first born, 16 per cent. were second born and the remaining number ranged from the third to the twelfth born. Of the colored infants, 46 per cent. were first born, 21 per cent. were second born, and the remaining number included those up to the tenth born.

All infants showing any suggestive manifestations of syphilis were examined serologically, and in eight instances postive Wassermann reactions were obtained, two in white and six in colored children. There was no apparent relation between these cases and those presenting either the least marked or most marked evidence of rickets.

Sex played no part in the incidence, or in the severity of the symptoms.

The climate of New Orleans is rather mild, there being only a very short period during which one is deprived of the benefits of being out doors. During the investigations, the winter was especially mild. Taking the attendance at the clinic as the index to the weather, the instinct of the mother determining whether the baby should be out or not, there was no definite curve, clearly showing that seasonal influence need not be considered.

From a study of the atmospheric conditions during the period of this investigation (Table 1), it will be noted from the percentage of possible sunshine that there was 3.9 per cent. more sunshine than the

TABLE 1.—*Monthly Meteorologic Summary*

	Possible Sunshine		Percentage of Temperature (F.)		Atmospheric Pressure			Precipitation (Inches)		Humidity			
						Since 1871				In Study		Normal	Average
	Average in Study	Average 1891-1916	Average in Study	Normal Average	Mean in Study	Highest	Lowest	Total per mo. in Study	Normal	7 a.m.	7 p.m.	7 a.m.	7 p.m.
October....	88	64	71.2	69.5	30.0	30.55	28.11	1.82	4.81	73	62	80	72
November..	64	55	66.6	60.6	30.1	30.68	29.47	3.83	3.79	85	72	82	74
December..	57	45	60.8	54.4	30.13	30.71	29.55	3.49	4.46	86	74	82	75
January...	36	48	51.0	53.0	30.14	30.80	29.27	5.22	4.63	86	81	82	74
February..	49	49	62.8	56.3	30.12	30.71	29.13	3.25	4.47	86	78	82	73
March.....	62	57	62.5	62.0	30.08	30.71	29.55	8.45	5.30	86	76	82	73
April.....	61	59	73.3	67.9	30.05	30.48	29.44	3.81	4.90	81	68	82	71
May.....	54	64	75.7	74.5	29.93	30.38	29.62	5.75	3.88	84	77	81	71
June.....	57	61	81.8	79.6	29.99	30.34	29.62	6.45	6.16	81	72	81	75
July.....	63	58	82.4	81.3	30.06	30.29	29.41	4.05	6.47	84	75	82	76
August.....	70	55	83.0	82.2	29.97	30.29	29.43	5.71	5.61	84	72	82	77
September.	70	61	80.8	79.2	30.00	30.31	28.11	0.93	4.81	83	71	82	75
Average for year....	60.9	57	70.9	69.2	30.05	30.52	28.39	52.79	57.02	83	73	82	74

normal average for the same period. The average temperature was 1.7 degrees (F.) higher for the year. The average atmospheric pressure reduced to sea level was 0.6 inch less than the average mean for the year. The total precipitation for the period of study was 4.23 inches less than the normal average, and the humidity was normal average. These facts are interesting as the amount of rickets in this study occurred with increased sunshine, increased temperature and diminished precipitation as compared with the normal average.

Of the 197 babies studied in all, there was at least one plus finding in the symptoms of rickets. These plus findings ranged from one in any one of the symptoms to a total of nine and one half in the combined symptoms in the white and ten in the combined symptoms in the colored. There were only three instances in this study in which there were one plus readings, two in the white and one in the colored. In each instance, the symptom thus noted was beading.

None of the individual clinical symptoms appeared to have a seasonal peak, but considered from the total peaks as reckoned from the greatest number of plus readings of all the clinical symptoms, there was a seasonal incidence of maximum findings as follows: The four weeks' period of maximum findings was from March 9 to March 29; six weeks' period, March 9 to April 13; eight weeks' period, February 9 to March 29; ten weeks' period, February 9 to April 13; twelve weeks' period, January 26 to April 13; fourteen weeks' period, January 12 to April 13; sixteen weeks' period, January 5 to April 20; eighteen weeks' period, December 29 to April 27; twenty weeks' period, December 29 to May 11; twenty-two weeks' period, December 29 to May 25; twenty-six weeks' period, December 8 to June 1. It will be seen that March was the month during which the seasonal manifestations of the disease was most marked. The disease was more marked

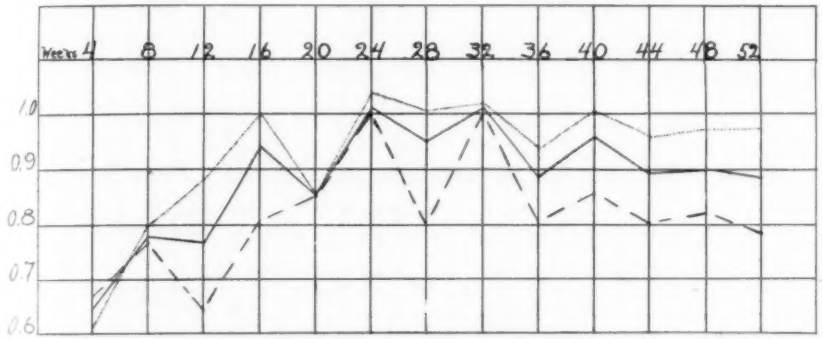

Chart 1.—Average intensity of symptom of beading throughout the first year. In this chart and the charts following, the broken line is the curve for white infants; the dotted line that for the colored infants; the solid line represents the average for the two races. The figures at the top represent the number of weeks and the figures at the left (Charts 1-6) the amount of plus reading.

during the half year from December 8 to June 1, the March peak being a little beyond the middle of the period.

The symptoms in the order of their frequency were beading, epiphysial enlargement, cranial bosses, flaring ribs, craniotabes, lateral flattening of the thorax and bow-legs. Lateral flattening was usually associated with the flaring ribs and was not especially studied separately. It was markedly evident in many instances. Bow-legs were noted in several instances but as this symptom could not be proved by exact methods, as for example, the roentgen ray, it was not gone into especially.

Beading was by far the most frequently met symptom, occurring in 97 per cent. of the white and in 98.9 per cent. of the colored. Chart 1 shows its severity according to age by weeks, both in the white and in

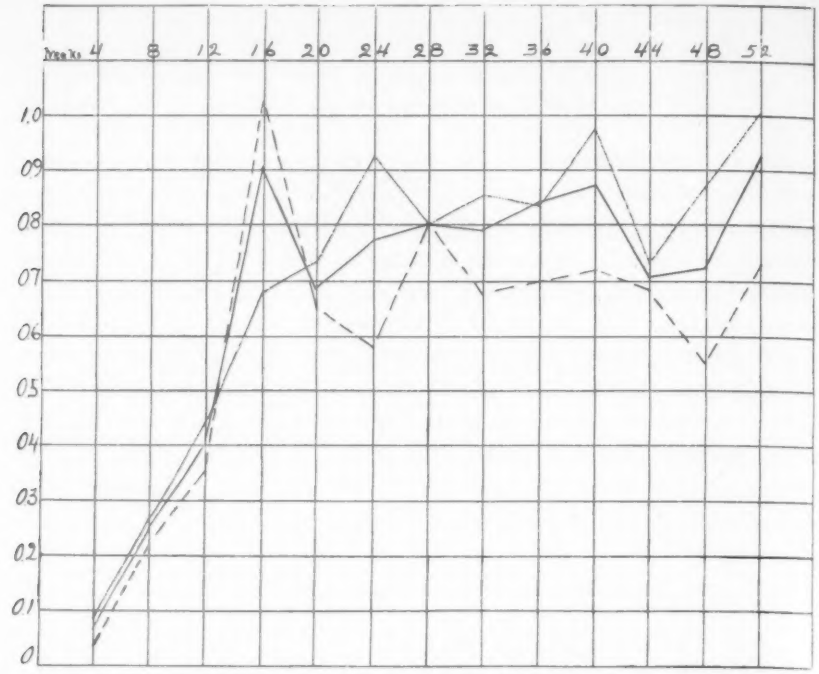

Chart 2.—Average intensity of symptom of enlarged epiphyses throughout the first year.

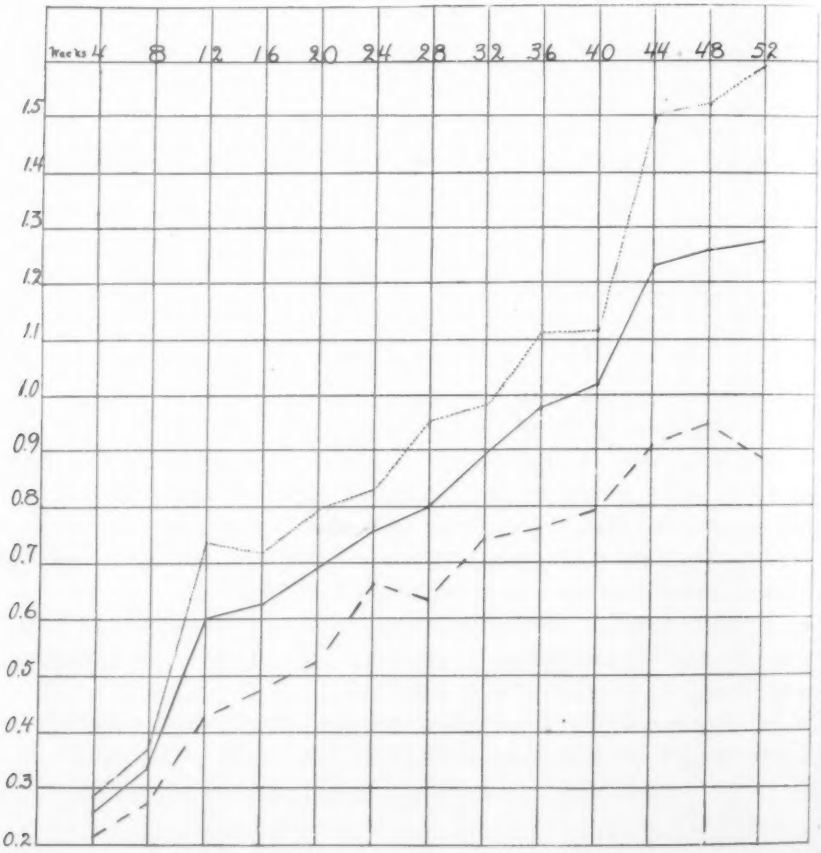

Chart 3.—Average intensity of symptom of cranial bosses throughout the first year.

the colored, and the average for the study. In the white, it was first noted as early as the fourth week, and it was also first noted as late as the thirty-second week. In the colored, it was also first noted as early as the fourth week, but it was first noted as late as the thirty-sixth week.

The beading curve shows a steady increase in incidence up to the end of the year.

Epiphysial enlargement is the symptom next in frequency, occurring in 84 per cent. of the white and in 87.1 per cent. of the colored. It was first noted as late as the thirty-sixth week, and as early as the fourth week in the white; and in the colored, it was first noted as late as the thirty-sixth week and as early as the fourth week. Chart 2 shows its severity according to age by weeks.

Cranial bosses showed an incidence of 68.63 per cent. in the white and 75.27 per cent. in the colored. It was first noted in both the white and colored at 4 weeks, and the latest age at which it was first noted in the white was 40 weeks and in the colored 32 weeks. This curve in Chart 3 shows a steady increase in the severity of the condition, with a rapid ascent toward the end of the year.

Flaring ribs showed an incidence of 62.58 per cent. in the white and 78.04 per cent. in the colored. In this symptom, the difference in its occurrence in the two races was greater than that of any of the symptoms, it being nearly 15 per cent. more frequent in the colored. It was first noted in both the white and the colored as early as the fourth week. The latest age at which it was first noted was 32 weeks in the white and 36 weeks in the colored. The curve in Chart 4 shows decidedly greater severity of this symptom in the colored over the white throughout the year.

Craniotabes, as shown in Chart 5, is extremely interesting. Its incidence of 33.33 per cent. in the white and of 43.48 per cent. in the colored is the least frequent of the principal symptoms. The curve of the severity of the symptoms, with its rapid ascent, short period of existence and its abrupt decline, is striking. It was first noted in both the white and colored at the age of 4 weeks, and was first noted as late as the sixteenth week in the white and twenty-fourth week in the colored.

It will be seen from the foregoing that each of the symptoms was first noted as early as the fourth week, and with the exception of craniotabes, was first noted as late as from the thirty-second to the fortieth week. The cranial bosses were the latest symptom to be first noted.

The relative occurrence of the symptoms in the races is also interesting. In the beading, the difference is only 1.9 per cent. This is the

most common symptom in both races, and its incidence is most nearly the same. The difference in the frequency of the individual symptoms in the white and colored increases as the frequency of the symptoms decreases, with the exception of craniotabes.

It is worthy of note that each symptom has a greater incidence in the colored, and in Chart 6, in which the severity of the combined

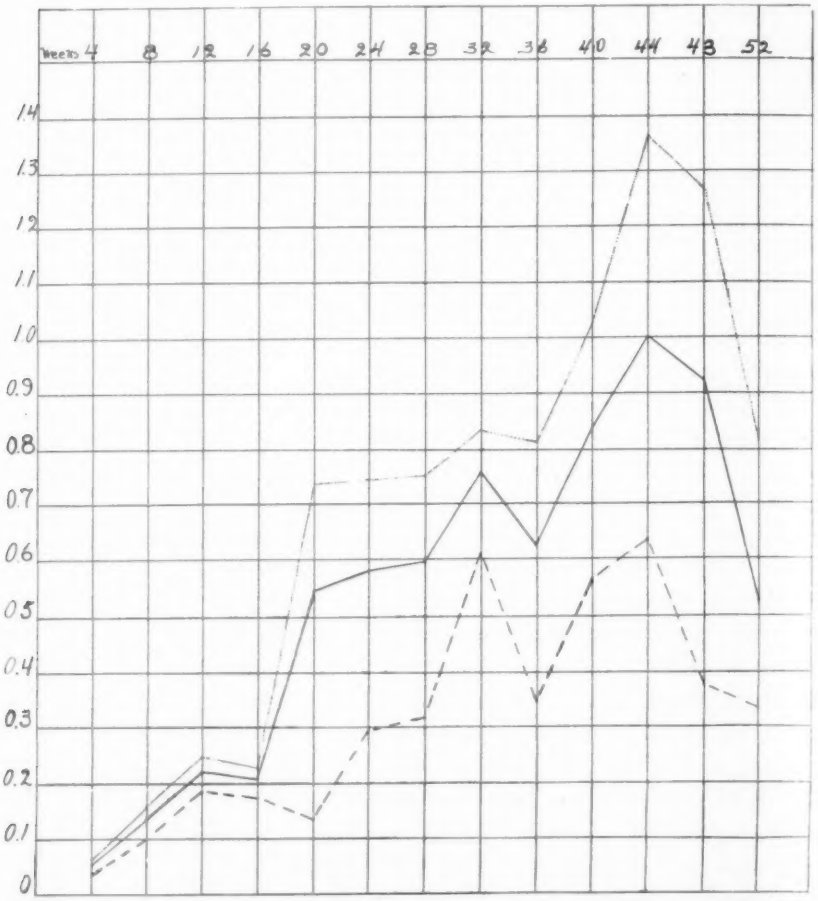

Chart 4.—Average intensity of symptom of flaring ribs throughout the first year.

symptoms is recorded, the curve is more even, with fewer variations in the curves of both the white and the colored, the curve of the colored being constantly above and that of the white always below the average for this study.

It is of especial interest to note how early (during the first three months) and rapidly rickets develops. It must not be looked on as a

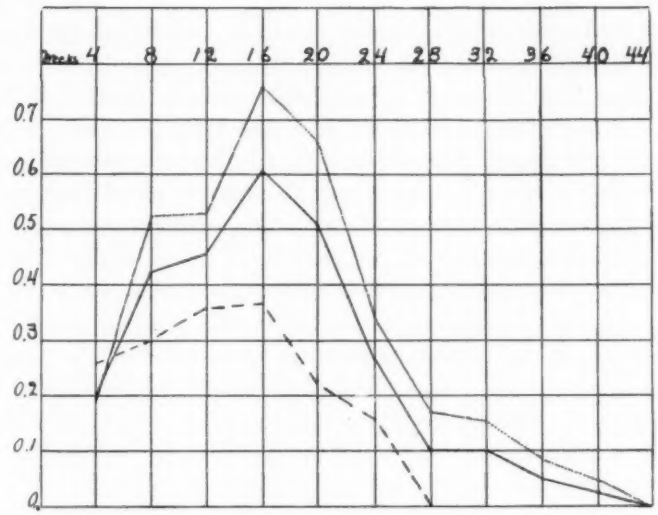

Chart 5.—Average intensity of symptom of craniotabes throughout the first year.

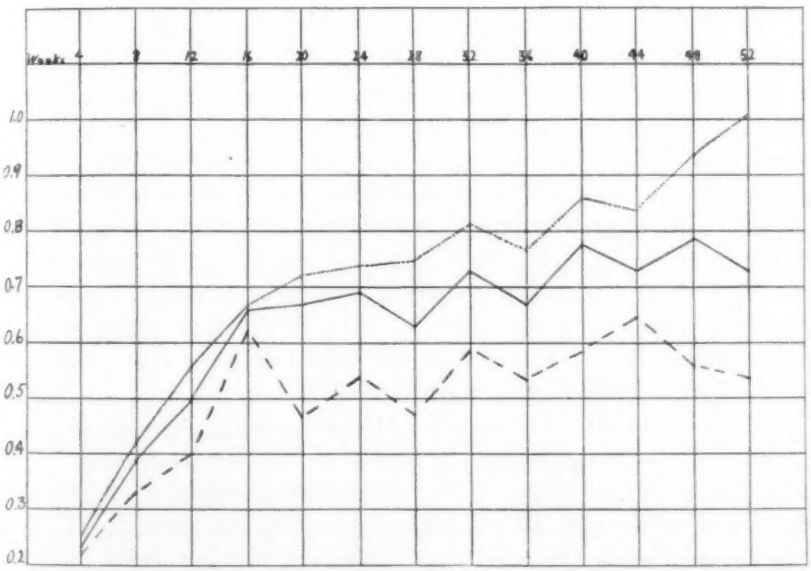

Chart 6.—Average intensity of combined clinical symptoms of rickets throughout the first year.

disease of the second half of the first year, but a disease which may begin shortly after birth.

The influence of rickets, as it occurs in the breast-fed, on the growth and development was also studied.

The average height of the white children was that of the normal average. The colored children were shorter than the white throughout the year. The height measurements in this study showed the most even curve of all the measurements. The most marked cases of rickets and the mildest cases were studied. Between the heights of these children and the general average, there were no noticeable differences.

The weight curve of the white children was a little above the normal average. In the colored, it was not satisfactory, its variations for the different ages being rather confusing, more particularly toward the latter part of the year. These variations may have been due to a lack of uniformity in feeding methods. The weights of the children suffering from marked rickets were below the normal average, while the weights of those with the mildest cases were about normal.

TABLE 2.—*Tissue Turgor (Percentage)*

	Excellent	Good	Fair	Poor
White	4	59.1	26.5	10
Colored	...	68.4	27.1	4
Average	1.4	65	27	6

The head measurements in the general average of the white in the study were a little greater at birth, but soon approximated the normal average and continued so thereafter. In the colored, the general average in early measurements was slightly greater but the increase throughout the year was below normal, so that at the end of the year the head was smaller than the normal average. The head measurements in the most severe and mildest cases of rickets in the white infants did not vary to any degree from the general average. In the cases of severe rickets in the colored, however, in the early months, there was a decided increase, and at the end of the year the measurement of the head was a half inch greater than the normal average.

The chests of the white infants in the general average were approximately normal. In the colored, at the end of the year, the chest measurement was less than normal. The differences in degree of severity of the rickets apparently did not cause any great variations in the chest measurements.

The measurements of the abdomen in the general average in the study in both white and colored infants were smaller than those of the chest. In the cases of marked rickets, the abdomens were larger

than the general averages, and in the mildest cases they were about the same for both white and colored.

Tissue turgor as an indication of the state of nutrition of the infants was noted in Table 2. It was found that in only 4 per cent. was it excellent, and these cases were all in white children. There were, however, more colored with good or fair tissue turgor than white, 68.4 and 27.1 per cent., respectively, as compared with 59.1 and 26.5 per cent. in the white. The grade of tissue turgor did not seem to be influenced by the severity of the rickets.

Dentition as shown in Chart 7 began a little earlier in the colored than in the white, the first tooth erupting at the twentieth week in the former and at the twenty-fourth week in the latter. The age of latest eruption of the first tooth in this study was 44 weeks in the white and 48 weeks in the colored, the average age of eruption of the first tooth being 32 weeks in the white and 33⅓ weeks in the colored. It will be seen from Chart 7 that the colored infants began to teethe earlier, and the teething curve rises steadily up to the forty-fourth week, when it passed that of the white infants. Teething in the white infants began four weeks later, assumed the lead at the twenty-fourth week and retained it up to the forty-fourth week. At the end of the year, the colored infants averaged one tooth more than the white.

In this study, the severity of the rickets did not seem to have any special influence on dentition, as shown in the comparison of the dentition in the most marked with the least marked cases of rickets, except that in the colored infants those with mildest rickets had more teeth, and at an earlier date. The influence of the severity of rickets on dentition is noted in the following tabulation:

DENTITION IN MARKED RICKETS

In the white, earliest eruption of first tooth, 28 weeks.
In the white, latest eruption of first tooth, 48 weeks.
In the white, average time eruption of first tooth, 38½ weeks.
In the colored, earliest eruption of first tooth, 28 weeks.
In the colored, latest eruption of first tooth, 52 weeks.
In the colored, average time of eruption of first tooth, 38½ weeks.

DENTITION IN LEAST RICKETS

In the white, earliest eruption of first tooth, 28 weeks.
In the white, latest eruption of first tooth, 52 weeks.
In the white, average time eruption of first tooth, 38½ weeks.
In the colored, earliest eruption of first tooth, 24 weeks.
In the colored, latest eruption of first tooth, 40 weeks.
In the colored, average time of eruption of first tooth, 29½ weeks.

There were no instances of abnormal teeth in this series.

Observations on the muscular development as shown by sitting alone, standing alone and walking alone did not reveal anything unusual.

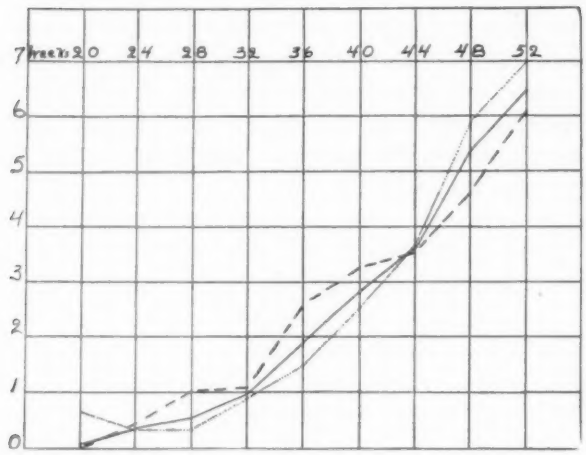

Chart 7.—Average number of teeth (figures to left of chart).

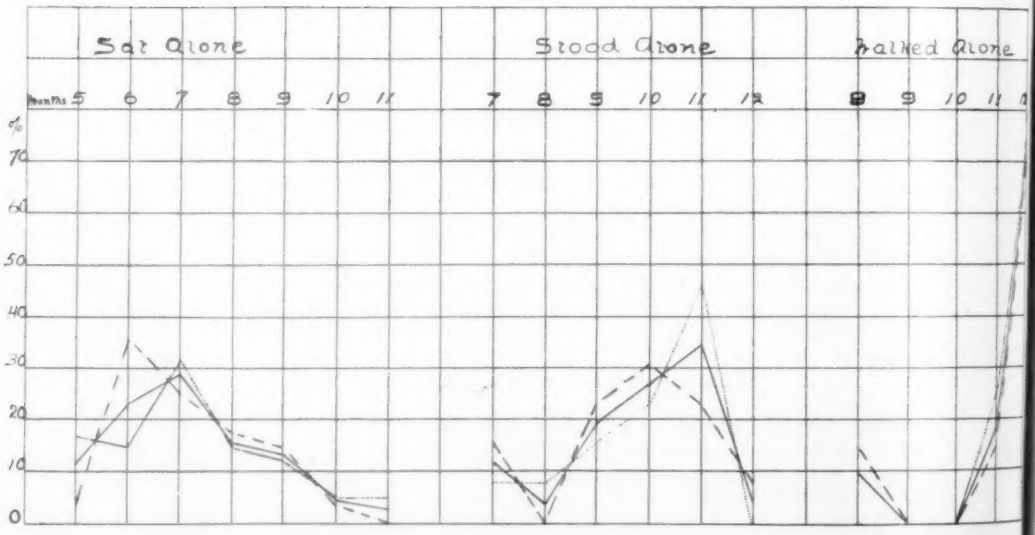

Chart 8.—Muscular development. The figures to the left represent the percentage of children.

In Chart 8, it will be noted that 35 per cent. of the white babies sat at 6 months, while only 31 per cent. of the colored could sit alone at 7 months. However, 17 per cent. of the colored babies sat alone at 5 months, while only 3½ per cent. of the white could do so. The greater number of white stood alone earlier than the colored—30.7 per cent. of the white stood alone at the tenth month, while 46 per cent. of the colored could stand alone at 11 months. Fifteen per cent. of the white were able to stand alone at 7 months, as compared with 7.6 per cent. of the colored.

The white infants began earlier to walk about, but the colored at the tenth month passed ahead of the white and remained in the lead thereafter.

SUMMARY

1. Only those cases in which the symptoms of rickets were noted before complemental feedings were instituted were included in this study.

2. Cod liver oil was not given in any case before there was unmistakable evidence of the existence of rickets.

3. Lack of supervision and care seemed to cause the rickets to develop with more intensity. There was a direct relation between the severity of the rickets and the interval of nonsupervision.

4. There was no noticeable increase in the symptoms of rickets after simple acute illnesses.

5. There was no apparent relation between the severity of the rickets and the numerical order of the child in a family, or the number of the children in a family.

6. Syphilis was noted in only eight instances as determined by the clinical manifestations of the disease and the Wassermann reaction. There was apparently no relation between syphilis and the severity of the rickets.

7. The atmospheric conditions during the period of this study showed that the amount of sunshine was greater and the temperature higher than the normal average; that the precipitation and atmospheric pressure were less than the normal average, and that the humidity was the normal average.

8. According to the clinical manifestations of rickets, the disease was present in every case in this study.

9. The disease was more marked in the colored subjects than in the white.

10. There was no seasonal peak for any of the individual symptoms. There was, however, a seasonal peak of the combined symptoms of the disease noted in the month of March.

11. Costal beading was the most frequent symptom. Enlarged epiphyses, cranial bosses, flaring ribs and craniotabes followed in the

order named. The short period of the existence of craniotabes, with its rapid increase and abrupt decline, was striking.

12. Each symptom was more common in the colored than in the white. Beading showed the least difference, i. e., 1.9 per cent. The differences increased as the frequency of the individual symptom decreased with the exception of craniotabes.

13. Rickets should not be looked on as a disease of the second half of the first year, but as a disease which may begin at least shortly after birth.

14. In the study of the influence of rickets on the growth and development of the child the following were the outstanding notations:

(*a*) The heights of the white babies were normal average, the heights of the colored were less.

(*b*) The weights of the white babies were a little above the normal average. The weights of the colored varied so much for the different ages that they were unsatisfactory for comparison. The weights of the markedly rachitic were below the normal average.

(*c*) While there were some slight variations in the measurements of the head, the most significant was the increased size of the markedly rachitic colored.

(*d*) The chests of the colored were smaller than the normal average.

(*e*) The abdomens in this study were smaller than the normal average. In the markedly rachitic, the abdomens were larger than the general average.

(*f*) Tissue turgor was recorded as excellent only in the white. It was noted as poor more frequently in the white than in the colored.

(*g*) Dentition began earlier in the colored, and at the end of the year the average number for the colored was greater than for the white. Aside from the fact that the colored with the mildest rickets had more teeth and had them earlier, nothing was noted in the study as to the influence of the severity of the rickets on dentition.

(*h*) A study of the muscular development as recokoned by the ability to sit alone, stand alone and walk alone showed some slight variations between the white and colored. It showed nothing of significance between the least marked and most marked cases of rickets.

REVIEW OF THE LITERATURE FOR 1921 AND 1922 ON NEUROLOGY OF CHILDREN *

AUGUST STRAUCH, M.D.

CHICAGO

ACUTE POLIOMYELITIS

Recent studies of Wahler [238] confirm the experience of others that the prognosis for the acute stage of poliomyelitis is favorable in the absence of symptoms of a lesion of the medulla oblongata. Very marked and extended paralyses at this stage as a rule become regressive and limited to definitely circumscribed foci. The prognosis for the post-acute stage as to restitution is doubtful. Those cases that were discharged with no or with insignificant improvement revealed practically no farther amelioration even with orthopedic and surgical treatment during the observation of from two to three years. In the cases showing a marked tendency toward improvement at the end of the acute stage, late reexamination revealed an additional, often striking, improvement, perhaps due to continued treatment. The prevention of deformities is of great importance, and children must be kept under observation during the entire growing period.

Paralysis affecting the upper extremities is milder and more amenable to improvement than that affecting the lower extremities. The improvement is most rapid in the first year, but under treatment may continue for four years, as the analysis of 180 cases by Lovett [239] demonstrates. In lower extremities, after the third year, there is a tendency to a slight loss of muscular power, partly due to deformities, an effort to prevent which must be made by every possible means.

Children with extensive paralysis following poliomyelitis develop certain types of locomotion. Magnus' [240] patient had acquired the locomotion of a quadruped with such agility that he presented a picture "not unlike that of a chimpanzee." In a very rare group, the afflicted become "hand-walkers." A unique case of this type, in an 11 year old boy, is reported by Kopits.[241] The lower extremities had been paralyzed since his second year. As two striking illustrations to the text demonstrate, the boy used the upper extremities as active support, for locomotion exclusively, maintaining his equilibrium either by "assuming an

* Received for publication, Oct. 6, 1923.

238. Wahler, F.: Arch. f. Kinderh. **69**:343 (May) 1921.

239. Lovett, R. W.: Third Year in Infantile Paralysis, J. A. M. A. **77**:1941 (Dec. 17) 1921.

240. Magnus, G.: Arch. f. Orthop. **19**:50, 1921.

241. Kopits, E.: Jahrb. f. Kinderh. **99**:268 (Sept.) 1922.

attitude resembling that of a standing bird" or by lifting his trunk into almost erect posture with the pelvis high up, from which the paralyzed legs dangle down. He displays a remarkable dexterity in playing ball, running and fighting, for instance.

Berghinz [242] encountered a girl who employed mirror writing or wrote normally at will with the left hand, following right hemiplegia from poliomyelitis; and Babonneix and Pollet [243] describe crossed localization of the paralysis involving the right arm and left leg, with marked changes in the bones and extreme hypotonia of the muscles.

The occurrence of four cases of poliomyelitis in the same family is reported by Mulsow and Matousek.[244] The source remained unknown.

As paralysis or weakness of the gluteal muscles, of the hip flexors and abdominal muscles, is of primary importance as a cause of lameness, operations on the thigh or the leg should be undertaken only, as Lovett [245] emphasizes, after careful study of the gait and with the understanding that the hip and abdominal lameness will not be affected materially by the procedure. The same author,[246] basing his conclusions as to diagnosis, prognosis and treatment on a study of 5,100 cases, emphasizes that early treatment consists of rest; that in the convalescent phase muscle fatigue is our chief danger and muscle reeducation our chief reliance, and that deformities prevent proper function and promote muscular deterioration.

The therapeutic procedure recommended by the Harvard Infantile Paralysis Commission, which consists chiefly in maintaining from the start the greatest rest and fixation obtained by recumbency, frames and splints, has been criticized by Feiss.[247] On physiologic principles, in order to prevent muscular deterioration by disuse, he urges early application of function by willed effort and reflex movements, the latter especially in infants.

The report of the Harvard Infantile Paralysis Commission published by Peabody,[248] reviewing the therapeutic results since 1916, fails to convince the Commission of the value of treatment in the preparalytic stage with intravenous injections of serum from patients who have recovered from this disease.

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242. Berghinz, G.: Pediatria **29**:301, 1922.

243. Babonneix and Pollet: Bull. et mém. Soc. méd. d. hôp. de Paris **45**:778 (May 27) 1921.

244. Muslow, F. W., and Matousek, W. J.: Family Outbreaks of Poliomyelitis, J. A. M. A. **76**:159 (Jan. 15) 1921.

245. Lovett, R. W.: Surg., Gynec. & Obst. **32**:20 (Jan.) 1921.

246. Lovett, R. W.: Diagnosis, Prognosis and Early Treatment of Poliomyelitis, J. A. M. A. **78**:1607 (May 27) 1922.

247. Feiss, H. O.: Treatment of Early Infantile Paralysis as Based on Physiologic Indications, J. A. M. A. **78**:85 (Jan. 14) 1922.

248. Peabody, F. W.: Boston M. & S. J. **185**:174 (Aug. 11) 1921.

According to Henderson [249] of Rochester, surgery is indicated only in residual paralysis, and only 25 per cent. of such patients can be helped by surgical procedures. The author discusses the various methods of dealing with the conditions by manipulations, tenotomies, arthrodeses, etc.

That nurses and physicians may transmit the virus, either by being affected themselves or by being healthy carriers, should be kept in mind. The experiences of Wallgren,[250] who himself has been a carrier, and the experiences of others cited by him warn anew of this possibility. As the disease is transmitted greatly by healthy carriers, home disinfection is no preventive measure, as pointed out by Mueller [251] in an article on the epidemiologic factors in the disease. Saunders [252] incriminates the fly *Lucilia Cesar* as playing a rôle in the propagation of the disease, maintaining that, in the absence of this insect, poliomyelitis is no more to be feared than malaria fever without anopheles.

ENDOGENOUS (HEREDITARY FAMILIAL) DISEASES OF THE NERVOUS SYSTEM AND THE MUSCLES

Familial Amaurotic Idiocy.—The studies of this disease by Marinesco [253] apparently sustain the assumption that the histopathologic changes in the brain are the expression of a disturbance of the activity of intracellular ferments, the swelling of the cells depending on hydrolysis produced by exaggerated activity of a protease.

The symptomatology and pathology of three atypical cases of Tay-Sachs' disease are minutely described by Torrence.[254]

Paterson [255] reports the occurrence of familial cerebral degeneration affecting eight children of a gentile family, beginning at the age of from 3 to 4 months and ending fatally. In the one case described, the diagnosis of familial amaurotic idiocy was made.

Friedreich's Ataxia.—This disease in two of five children in a full blooded negro family, a very rare occurrence in this race, was observed by Lloyd and Newcomer,[256] and Hess [257] saw the disease in twins.

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249. Henderson, M. S.: Surgery in Infantile Paralysis, J. A. M. A. **79**:1714 (Nov. 11) 1922.

250. Wallgren, A.: Upsala Läkaref. Förh. **27**:45, 1922; abstr., Zentralbl. f. Kinderh. **14**:539, 1923.

251. Mueller: Deutsch. med. Wchnschr. **48**:1569, 1922.

252. Saunders, E. W.: Missouri M. A. J. **18**:160 (May) 1921.

253. Marinesco, G.: Encéphale **16**:561 (Dec.) 1921; abstr., J. A. M. A. **78**:688 (March 4) 1922.

254. Torrence, H. W.: Glasgow M. J. **15**:341 (June) 1922; abstr., J. A. M. A. **79**:504 (Aug. 5) 1922.

255. Paterson, D.: Proc. Roy. Soc. Med. **15**:46, 1922.

256. Lloyd, J. H., and Newcomer, H. S.: Friedreich's Ataxia, Arch. Neurol. & Psychiat. **6**:157 (Aug.) 1921.

257. Hess, J. H.: M. Clinics N. America **5**:1749 (May) 1922.

Ataxia symptoms of the tabetic-spastic-cerebellar type, as described by Oppenheim, Concetti, Taylor, Gordon and others, were recorded by Orrico[258] in two boys of five, since they began walking. Discussing the similarity to and difference from the syndromes of Friedreich and P. Marie, the author insists that these clinical pictures should be distinguished from both types. The affection involves the posterior cerebellar tracts and the pyramides.

Bergmann[259] reports three Swedish agricultural families with Friedreich's ataxia and one with Marie's type. In one of the families, six children of fifteen, and in another four of eleven, presented the anomaly.

That not only a familial factor but also other pathogenetic elements, such as cerebrospinal processes due to syphilis or inflammations of other infections, can play a rôle in the causation of Friedreich's syndrome is again substantiated by the observation by Urechia and S. Mihalescu[260] of this symptom complex in a boy, due to acquired syphilis; and Linder[261] cites congenital syphilis under the picture of hereditary ataxia, presenting a rare combination of Friedreich's and Marie's type in a girl 6½ years old. This case, as that of Allen Star, is unique in the medical literature.

The relationship of congenital Friedreich's disease to other heredodegenerative affections is obvious in the association of the syndrome with nuclear paralysis of ocular muscles and with chorioditis disseminata observed by Schob.[262] Typical cerebellar symptoms were combined with an affection of the posterior spinal tracts and kyphosis. Remarkable was the presence of the same eye disturbances in the sister, the lack of progression and the absence of any hereditary or familial character except for the eye disturbances.

In one of the cases of Sanchis Banús and S. Gonzáles Taez (Arch. neurobiol. **3**:43, 1922, Friedreich's symptom complex was combined with progressive cerebellar dissynergia; in the other, the latter condition existed in its pure form.

Progressive Myopathia.—Four brothers under 13 years of age affected with primary progressive myopathia and presenting different stages of the disease are the subject of a report by Auricchio.[263]

Duken and Weingartner[264] describe progressive spinal atrophy of Werdnig-Hoffmann type, in a boy 4½ months old, beginning probably in

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258. Orrico, J.: Arch. latino-am. de pediat. **16**:799, 1922.
259. Bergmann, E.: Upsala Läkaref. För. **26**:1, 1921.
260. Urechia, C. L., and Mihalescu, S.: Ztschr. f. ges. Neurol. u. Psychiat. **71**:207, 1921.
261. Linder, C.: Jahrb. f. Kinderh. **100**:65, 1922.
262. Schob, F.: Ztschr. f. ges. Neurol. u. Psychiat. **73**:188, 1921.
263. Auricchio, L.: Pediatria **30**:488 (June 1) 1922.
264. Duken, J., and Weingartner, A.: Ztschr. f. Kinderh. **29**:245, 1921.

intrauterine life. A rare symptom in this affection, paradoxic thoracic respiration due to paralysis of the musculature of the thorax, was of special interest. Postmortem examination revealed, in addition to degeneration of the ganglion cells of the facial nerve and other nuclei, of the anterior horns, and of Clarke's columns, cavities in the entire spinal cord formed by dilated capillary lymph spaces or empty ganglion cell beds, and heteropias of white substance and ganglion cells. The author sees the basis of the affection in a constitutionally substandard abiotrophic central nervous system with extremely lowered resistance for functional tasks.

Thomsen's Disease.—This disease existing in three generations is analyzed in great detail by Rosett.[265]

Periodic Myoplegia.—Neustädter [266] gives the interesting history of a family with several members suffering from periodic paralysis or insanity, and reports a case in full detail. This patient, 18½ years old, had since the age of 14 periodic paroxysms of marked weakness involving all the extremities, the trunk and neck, lasting from night till afternoon and being associated with fatigue, vomiting, headache and perspiration.

PERIPHERAL NERVES

Among forty-five cases of obstetrical palsies of the brachial plexus seen at the Mayo Clinic, Adson [267] found 73 per cent. in association with forceps delivery. Recovery to some degree occurred in practically every case. The greatest amount of recovery was experienced during the first three months. In some cases, improvement had continued over a period of twelve years. In the patients under 2 years of age, there was approximately one-third return of function; in those under 5 years, one-half return of function, and in those over 5 years the improvement progressed to about two-thirds normal function. Dislocations of the shoulder encountered seemed to develop rather after than during birth. That even total brachial paralysis at birth heals, generally without any disturbance, becomes evident from the publication of Froelich.[268]

Friedberg [269] recalls that chronic saturnism in childhood differs from that in the adult in that development is slower, colic is rare; characteristic blood findings are lacking, as is the gray gingival margin and there is an atypical localization of motor disturbances in the lower extremities. The difficulty of diagnosis is borne out by a case cited that

265. Rosett, J.: Brain **45**:1 (June) 1922.
266. Neustädter, M.: New York J. Med. **22**:65 (Feb.) 1922.
267. Adson, A. W.: Northwest Med. **21**:33 (Feb.) 1922.
268. Froelich: Rev. de chir. **59**:419, 1921.
269. Friedberg, E.: Arch. f. Kinderh. **71**:25 (Feb.) 1922.

had escaped recognition for several years. With the elimination of the cause—playing with lead toys and lead types for years—the disturbance vanished.

Postdiphtheritic paralysis of the right pterygoid muscle and marked involvement of the second and third branches of the trigeminus nerve with recuperation have been reported by Huenerberger.[270] In a second patient, the muscles of neck and back were involved, the clinical picture resembling poliomyelitis. Labbe [271] was able to cure tardy diphtheritic paralysis by systematic treatment with antitoxin in seven children. In one child, the paralysis of the soft palate had been of four years' standing. Hallez [272] found lymphocytosis, hyperalbuminosis and hyperglycorachia in the cerebrospinal fluid of a girl of 12 suffering from postdiphtheritic paralysis.

Certain aspects of postdiphtheritic diaphragmatic paralysis are described by Mixsell and Giddings,[273] who report eight fatal cases occurring among 4,259 instances of diphtheria. In cases presenting involvement of the diaphragm, the mortality has been 100 per cent.

In a paper on infantile beriberi in the Philippines, Tupas [274] reports thirty-six cases, thirty of the patients being under 3 months old, the youngest 14 days, the oldest 1 year and 5 days old. The mortality was 30.5 per cent. The lesions in five cases examined postmortem were marked dilatation of heart, hydropericardium, punctate hemorrhages of lungs, congestion in various viscera and degenerative neuritis of the vagus nerve.

Erythroedema.—The disease described first in Australia by Swift (1914) in children as erythroedema, by Snowball as "raw beef hands and feet," and known in Sidney as "pink disease," has been observed repeatedly in this country, where the name acrodynia, as suggested by Weston (1920) is frequently used. Brown and Courtney with MacLachlan [275] discuss at length the complex clinical picture of this affection, which is considered a form of polyneuritis or radiculitis. The chief features are: nervous disturbances of sensory and trophic character; distressing paresthesias in fingers, feet and toes, leading to incessant scratching and rubbing; in some cases, diminished or absent cutaneous sensibility to pin pricks, and photophobia. The skin lesions are of greater value in the diagnosis, consisting in an erythematous eruption of

270. Huenerberger, H.: Arch. f. Kinderh. **71**:175, 1922.
271. Labbe, R.: Arch. de med. enf. **24**:612 (Oct.) 1921.
272. Hallez: Presse méd. **29**:878, 1921.
273. Mixsell, H. R., and Giddings, E.: Certain Aspects of Postdiphtheric Diaphragmatic Paralysis, J. A. M. A. **77**:590 (Aug. 20) 1921.
274. Tupas, A. V.: Philippine Islands M. A. J., 1921, p. 108.
275. Brown, A.; Courtney, A. M., and MacLachlan, I. F.: Arch. Pediat. **38**:609 (Oct.) 1921.

almost edematous appearance, involving fingers and toes, and frequently extending to wrists and ankles. On the palms and soles, there is desquamation of "pin prick" character. There is excessive perspiration, and occasionally loosening and falling out of the teeth. Dulness and apathy alternate with restlessness. Anorexia and emaciation are present. Polymorphonucleosis is a symptom. There is a constant nasopharyngitis, on the subsidence of which, especially on removal of tonsils and adenoids, the children begin to improve. The seasonal incidence is suggestive of respiratory infections, including influenza as an etiologic factor, the syndrome probably being due to toxins. Eight observations are reported and the differential diagnosis is discussed.

As Wood [276] has notes on eighty-eight cases, the disease seems to be not uncommon in Australia.

Zahorsky [277] pleads for the adoption of the original term erythredema as used by the Australian physicians, especially since there is no identity with the great epidemics of acrodynia in Paris at the beginning of the last century. He observed pronounced atrophy of the muscles in one of his seven cases. In another, there were large areas of anesthesia; and in two babies, voluntary movements of both lower extremities ceased completely for several weeks. Field [278] presents a typical case in an 18 months old baby and states that about 100 cases have been reported to date. Mutilations and ulcerations, namely loss of finger tips due to gangrene, and chronic ulcers in each sole, from which, as from the nasal discharge, a diphtheroid bacillus was cultivated, have been reported by Weber.[279] Thursfield and Paterson [280] describe a case under the title dermatopolyneuritis, presenting signs of neuromuscular lesions with extreme hypotonia. An example is cited by Miller [281] under the name of "polyneuritic syndrome," and a doubtful case in which bronchiectasis is credited with the same relation to erythrodema as the nasal infections in some of the reported cases is described by Davis.[282]

Vipond,[283] from the study of two instances, concludes that the nose cavity is the point of inoculation, as a discharge is the first sign. Then follows general adenopathy pointing to a generalized infection. The next symptoms to be observed are the characteristic findings on hands

276. Wood, A. J.: M. J. Australia **1**:145 (Feb. 19) 1921.
277. Zahorsky, J.: Semeiology of Pink Disease (Erythredema, Acrodynia), J. A. M. A. **79**:1975 (Dec. 9) 1922; Missouri M. A. J. **19**:296 (July) 1922.
278. Field, M. C.: Arch. Pediat. **39**:116 (Feb.) 1922.
279. Weber, F. P.: Brit. J. Dis. Child. **19**:17 (Jan.-March) 1922.
280. Thursfield, J. H., and Paterson, D. H.: Brit. J. Dis. Child. **19**:27 (Jan.-March) 1922.
281. Miller, M. K.: Indiana M. A. J. **14**:144 (May) 1921.
282. Davis, Clara M.: Arch. Pediat. **39**:611 (Sept.) 1922.
283. Vipond, A. E.: Arch. Pediat. **39**:699 (Nov.) 1922.

and feet, these being of vasomotor nature. He obtained a gram-positive diplococcus from the material aspirated from an enlarged lymph gland. Vaccine prepared from this micro-organism had in both instances splendid results.

SPASMOPHILIA; INFANTILE TETANY

Nassau [284] distinguishes two different types of tetany. 1. The early form, which occûrs in the first three months. It is initiated by eclamptic attacks and other signs, such as the positive facial phenomenon. Kathodic overirritability may follow after an interval of up to 18 days. 2. The later form, which occurs in the second semester of life and is characterized by the frequency of laryngospasm as the initial symptom of manifest tetany. A predisposition for edema seems to be characteristic of spasmophilia and may occur in both types. In the eclamptic form, rapidily passing changes, oscillations in the "milieu interne" seem to play an important rôle, and in the laryngospastic form, possibly avitaminosis. The disturbance of salt metabolism is the common symptom; in the early form, an exaggerated retention of sodium and potassium in contrast to calcium, in the later type, a decreased retention of calcium being present.

A horse shoe shaped depression of the cornea during eclamptic attacks was observed by Ochsenius,[285] possibly caused by reduction of the intra-ocular tension.

Lemair and Olivier [286] aver that tetany in the infant may cause gastrospasm or pylorospasm, manifesting itself in habitual vomiting, which yields rapidly to the classic antitetany treatment.

Moschini [287] describes three cases of bronchotetany with necropsy findings. The importance of the recognition of this condition is evident, as antitetany measures would attenuate or cure bronchotetany.

The view expressed by Escherich and Ibrahim that the cause of sudden death among spasmophilic children is heart tetany lately found support in Koplik's [288] observations of bradycardia during attacks of sudden apnea. The author declares this symptom pathognomonic of spasmophilia. On the other hand, in the case reported by Iwabuchi,[289] marked pathologic changes in the vagus nucleus are described as the possible cause of death.

Among 1,648 schoolchildren examined by Schulze,[290] Chvostek's sign was present in 139 cases. As more than two-thirds of these were

284. Nassau, E.: Ztschr. f. Kinderh. **28**:310 (April) 1921.
285. Ochsenius, K.: Monatschr. f. Kinderh. **21**:151 (May) 1921.
286. Lemair, H., and Olivier, M.: Nourisson **10**:305 (Sept.) 1922.
287. Moschini, C.: Policlinico (sez. prat.) **28**:1616 (Nov. 28) 1921.
288. Koplik, H.: Arch. Pediat. **39**:504 (Aug.) 1922.
289. Iwabuchi, T.: Ztschr. f. Kinderh. **33**:223 (Aug.) 1922.
290. Schulze, A.: Monatschr. f. Kinderh. **22**:484, 1921.

to be considered as spasmophilic, the author concurs in the opinion of those who declare the facial phenomenon at this age at least as extremely suspicious of latent spasmophilia. Mosse,[291] on the basis of clinical observations and the fact that his determinations of calcium, phosphorus and the bicarbonate of blood serum revealed no differences between the groups of children with and without facial sign, comes to the opposite conclusion.

Myographic curves taken by Bossert and Gralka [292] in twenty-two children suffering from various signs of spasmophilia are interpreted as indicating that in infantile tetany (in the strict sense) calcium deficiency, possibly due to dysfunction of the parathyroids, entails imbibition of the musculature, altering its contractibility and resulting in carpopedal spasms, and that in the group of babies with laryngospasm as the cardinal symptom there exists an extreme overirritability of the nervous system referable to a prevalent calcium deficiency in the nerve tissue; that in eclampsia it is the prevalence of calcium deficiency in the brain especially that is to be held the responsible factor.

The influence of sodium and potassium phosphates on normal and spasmophilic infants has been studied by a number of authors. In numerous experiments conducted by Jeppsson,[293] administration of kalium and natrium phosphate provoked increased galvanic and mechanic irritability, typical laryngospasm and, in one child, typical tetany. In one experiment, dangerous expiratory apnea appeared. In dogs and rabbits, galvanic and mechanical overexcitability, eclamptic convulsions and tetany spasms and respiratory spasms, mostly of the character of laryngospasm, could be produced. The effects of the alkali phosphates was not due exclusively to the one or the other of their ions. Whey freed of its phosphates failed to increase the irritability.

Artificial mixtures of the salts approximately as they are present in cow's milk increased the tendency to spasmophilia, in the experiments of Wernstedt,[294] by disturbing the salt metabolism, and Roemer [295] demonstrated the tetanigenous effect of the basic sodium phosphate. On the other hand, Calvin and Borovsky [296] were unable to obtain the results of Jeppsson; and Wetzel [297] is convinced that the kalium ion as

291. Mosse, K.: Jahrb. f. Kinderh. **99**:244 (Sept.) 1922.
292. Bossert, O., and Gralka, R.: Jahrb. f. Kinderh. **94**:145, 1921.
293. Jeppsson, K., and Klercher, K. O.: Ztschr. f. Kinderh. **28**:71-167 (March 21) 1921.
294. Wernstedt, J. W.: Acta Pediat. **1**:133 (June) 1921; **1**:257 (Dec.) 1921.
295. Roemer, P.: J. de med. de Paris **41**:647, 669, 1922.
296. Calvin, J. K., and Borovsky, M. P.: Results Following Administration of Alkali Phosphates to Spasmophilic Rachitic and Normal Infants, Am. J. Dis. Child. **23**:238 (March) 1922.
297. Wetzel, A.: Ztschr. f. Kinderh. **32**:105 (March) 1922.

such has no spasmogenic action and that the untoward effect of cow's milk on spasmophilia is not to be ascribed to the larger amount of kalium ions in comparison to that in human milk.

The association of tetany with lowered calcium in the blood has received attention by Kramer, Tisdall and Howland,[298] who regularly found the calcium of the serum diminished in active tetany. They conclude that the increased irritability is due to the diminished concentration of calcium in the serum or in the ratio $\frac{Ca + Mg}{Na + K}$, the stimulating effect of the sodium and potassium being unopposed by the inhibitory effect of calcium. Thiemich's sign in older children, however, according to von Meysenburg,[299] is not based on lowered blood calcium.

That hyperphosphemia is an important symptom, possibly one of the causative factors in tetany, is believed by Elias and Spiegel,[300] who found the inorganic phosphorus content in the serum characteristically increased in tetany.

A number of authors assume that alkalosis is the underlying factor in tetany; and recently it was discovered that guanidin may provoke the syndrome without any relationship to calcium deficiency. Grant [301] records symptoms of tetany following an overdosage of sodium bicarbonate, which upset the ratio of sodium bicarbonate to carbonic acid in the blood. Tetany in gastric obstruction is declared to be due to an excess of sodium bicarbonate, resulting from the fact that it is impossible for the secreted hydrochloric acid to pass into the intestines to stimulate the flow of alkaline intestinal secretion. Goldmann [302] again reports eleven cases of bicarbonate tetany due to hyperpnea, with consequential alkalosis through excessive reduction of carbon dioxid. In the cases of Barker and Sprunt [303] and of Haas,[304] the tetany attacks were caused by hyperpnea in epidemic encephalitis. Freudenberg and György [305] come to the conclusion that the alkalosis existing in manifest tetany acts by altering the blood content of active calcium (dissociation of blood calcium), and that guanidin acts by displacing the calcium in the colloids. Consequently, they treat active tetany with "acidotic" remedies, that

298. Kramer, Benjamin; Tisdall, F. F., and Howland, James: Infantile Tetany, Am. J. Dis. Child. **22**:431 (Nov.) 1921.

299. Von Meysenburg, L.: Studies in Spasmophilia; Blood Calcium and Calcium Therapy in Older Children with Thiemich's Sign, Am. J. Dis. Child. **21**:150 (Aug.) 1921.

300. Elias, H., and Spiegel, E. A.: Wien. Arch. f. inn. Med. **2**:447 (June) 1921.

301. Grant, S. B.: Tetany, Arch. Int. Med. **30**:305 (Sept.) 1922.

302. Goldmann, Alfred: Clinical Tetany by Forced Respiration, J. A. M. A. **78**:1193 (April 22) 1922.

303. Barker, L. F., and Sprunt, T. P.: Endocrinology **6**:1 (Jan.) 1922.

304. Haas, S. V.: Arch. Pediat. **38**:785, 1921.

305. Freudenberg, E., and György, P.: Klin. Wchnschr. **1**:222 (Jan. 28), 410 (Feb. 25) 1922; Jahrb. f. Kinderh. **96**:5, 1921.

in the metabolism tend to increase acidity, such as hydrochloric acid milk, introduced by Scheer,[306] and "salmiak" (ammonium-chlorid), which Haldane[307] proved to produce in man marked and prolonged acidosis by lessening the alkali reserve. The results were excellent, apparently proving the correctness of the premises. However, Jones and Nye[308] and Calvin and Borovsky[309] dispute the theory of alkalosis in tetany.

Coda[310] and other authors incriminate the endocrine system, which may act on the vegetative nervous system, the calcium metabolism, the so-called myoneural conjunction or all these factors.

As regards the pathogenesis of the type of tetany following parathyroidectomy, the experiments made on dogs by Dragstedt[311] and Luckhardt and Rosenbloom[312] indicate that it is due to toxic substances chiefly derived from the gastro-intestinal tract through the activity of proteolytic bacteria, most probably of the nature of amins. The function of the parathyroids is to prevent this intoxication; and dogs may be kept alive indefinitely after parathyroidectomy, if by certain means this toxemia of intestinal origin is prevented.

The investigations of Hess[313] of New York with Gutman, and of Hess and Unger[314] demonstrated that, in infants, the inorganic phosphorus of blood can be raised to the normal level by exposures to the sun's rays or to the carbon arc lamp. This fact and the marked seasonal variations in the sun's spectrum prompted investigations by Hess and Lundagen,[315] which demonstrated a seasonal tide of blood phosphate during the spring and summer, the dominating factor being the sun light. Hess associated these facts with the peak of tetany and with rachitis.

GENERAL CONVULSIONS

That there exists a predisposition to convulsions separate from spasmophilia, that eclampsia and tetany have not the close relationship generally assumed is the contention of Goett and Wildbrett,[316] who

306. Scheer, K.: Jahrb. f. Kinderh. **97**:130 (Feb.) 1922.
307. Haldane, J. B. S.: J. Physiol. **55**:265 (Aug.) 1921.
308. Jones, M. R., and Nye, L. L.: J. Biol. Chem. **47**:321 (July) 1921.
309. Calvin, J. K., and Borovsky, M. P.: Spasmophilia and Alkali Reserve, Am. J. Dis. Child. **23**:493 (June) 1922.
310. Coda, M.: Pediatria **30**:764 (Aug. 15) 1922.
311. Dragstedt, L. R.: Pathogenesis of Parathyroid Tetany, J. A. M. A. **79**:1593 (Nov. 4) 1922.
312. Luckhardt, A. B., and Rosenbloom, P. J.: Science **56** (July 14) 1922; Proc. Soc. Exper. Biol. & Med. **19**:129, 1921.
313. Hess, A. E., and Gutman, P.: Proc. Soc. Exper. Biol. & Med. **19**:31, 1921.
314. Hess, A. F., and Unger, L. J.: Use of Carbon Arc Light in Prevention and Cure of Rickets, J. A. M. A. **78**:1596 (May 27) 1922.
315. Hess, A. F., and Lundagen, M. A.: Seasonal Tide of Blood Phosphate, J. A. M. A. **79**:2210 (Dec. 30) 1922.
316. Goett and Wildbrett: München. med. Wchnschr. **69**:884, 1922.

found that babies of families in whom there exist lefthandedness, various spasms or genuine epilepsy are predisposed to eclampsia.

An interesting problem as to the pathogenesis of convulsions in early childhood is involved in the observation of Reiche [317] of an apparently primarily increased chlorid concentration in the cerebrospinal fluid, causing, according to the law of osmosis, an increase of the quantity of the spinal fluid, and thus also of the intraspinal pressure, with consequent convulsions, in a baby, 1 year old.

The importance of reflex irritation as a cause of convulsions has been undoubtedly exaggerated. Morse [318] never saw convulsions due to phimosis, eye strain, teething or worms, etc.; with the exception of one case in which convulsions ceased after administration of an anthelmintic, and another case in which the seizures ceased after stretching of the sphincter ani by a quack doctor. However, in convulsions following eating of indigestible and improper food, reflex irritation seems more important than toxic absorption.

The treatment of convulsions, especially of spasmophilic nature, has been outlined by Blühdorn.[319] That epilepsy as a disease is not transmitted from parent to offspring but rather indicates a nervous system lacking in normal stability is the belief of Thom and Walker.[320] The manifestations of this instability may be mental deficiency, insanity, neurologic and psychopathic disorders and convulsions from various exciting causes that would have little or no effect on a normal nervous system. The statistics of Burr [321] point likewise to an indirect, general, nonspecific inheritance.

Antheaume and Trepsat [322] produced violent convulsions, followed by death, in several rabbits by injecting the serum of an epileptic.

That jacksonian epilepsy in a large percentage of cases does not mean a definite focal disease of the cortex is emphasized by Wilson,[323] who enumerates various conditions, some remote from the cortex, that are occasionally associated with monospasms or hemispasms. Probably, the commonest cause of this form of spasm is idiopathic epilepsy itself ("idiopathic epilepsy with inside jacksonian spasm"). A person with this type should, therefore, not be operated on unless other symptoms of intracranial disease are present.

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317. Reiche, F.: München. med. Wchnschr. **68**:202, 1922.
318. Morse, J. L.: Convulsive Disorders of Childhood, J. A. M. A. **78**:175 (Jan. 21) 1922.
319. Blühdorn, K.: Med. Klin. **18**:733 (June 4) 1922.
320. Thom, D. A., and Walker, G. S.: Am. J. Psychiat. **1**:613 (April) 1922.
321. Burr, C. W.: Heredity in Epilepsy, Arch. Neurol. & Psychiat. **7**:721 (June) 1922.
322. Antheaume, A., and Trepsat, L.: Encéphale **17**:103 (June) 1922.
323. Wilson, George: Diagnostic Significance of Jacksonian Epilepsy, J. A. M. A. **76**:842 (March 26) 1921.

Jacksonian epilepsy, with attacks in the left arm, in a girl of 4 was completely cured following treatment for ascaris. Sironi,[324] who reports this case, theorizes that the toxins of the helminths may have acted on a focus of lesser resistance, perhaps a residue of a previous infection in the brain.

The investigations of reflex epilepsy found a real basis in the striking results of animal experiments conducted by Amantea,[325] who, by cutaneous stimulations, especially in the so-called reflexogenic areas, succeeded in eliciting epileptic seizures.

Theories as to the pathogenic relation of the so-called pyknoleptic attacks or Friedmann's disease are dealt with by Meyer.[326] These "repeated minor seizures" resemble petit mal in some respect, but not in others, as the children remain mentally normal; ordinary antiepileptic measures have no effect; the attacks cease as a rule toward puberty, and the prognosis is favorable. During an attack, there is a sudden and short suspension of the higher psychic functions, a sudden interruption of the occupation and rolling of the eyes. After from one-quarter to one-half minute, the attack subsides, and the occupation is continued. He reports four cases, in children from 7 to 12 years of age with an inherited nervous taint, but bright and lively. The seizures occurred up to thirty times a day, lasting from one-quarter to one-half minute. While some authors consider the affection a form of epilepsy with intact mentality, Meyer classifies it as a special group of the neuropathic forms of reactions, entirely apart from epilepsy or hysteria. Physical fatigue seemed to elicit the manifestations. He recommends calcium up to 6 gm. a day, with small doses of phenobarbital (luminal).

Stargardter,[327] who describes the pictures of pyknolepsy in four children, and Husler,[328] who presents a monograph on the clinic of epileptiform affections in children, see the pathogenic basis of these "multiple absences" in marked neuropsychopathy. On this basis, these simple and uniform seizures develop together with other neuropathic manifestations. One of the cases of Stargardter demonstrates that pyknolepsy may grow also on the basis of true epilepsy. Caffein proved efficacious in the treatment.

Deep unconsciousness, spasms, nocturnal character, falling with injuries, the onset of the attacks before the fifth year of life, exhaustion and the therapeutic effect of phenobarbital would, in the experience of Stier,[329] render the seizures suspicious of being true epilepsy and thus make the prognosis more serious.

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324. Sironi, L.: Pediatria **29**:397 (May 1) 1921.
325. Amantea, G.: Arch. f. d. ges. Physiol. **188**:287, 1921; Policlinico (sez. prat.) **27**:462, 1920.
326. Meyer, M.: Ztschr. f. Kinderh. **27**:293 (Jan.) 1921.
327. Stargardter, J.: Jahrb. f. Kinderh. **95**:230, 1921.
328. Husler, J.: Ergebn. d. inn. Med. u. Kinderh. **19**:652, 1921.
329. Stier, E.: Ztschr. f. ges. Neurol. u. Psychiat. **80**:143, 1922.

Tests of the oculocardiac reflex, made by Rubinovitch and Chavany,[330] in fifty-eight epileptics, revealed that in 62.5 per cent. of the cases, the bradycardia was followed by tachycardia for a minute. They see in this fact a new means for the differential diagnosis in doubtful cases.

Phenobarbital or sodium phenobarbital has found a widespread therapeutic use in epilepsy, and is considered by most authors the most effective symptomatic remedy, reducing decidedly the number and severity of the convulsions. No mental deterioration nor any toxic or accumulative by-effects were observed by Grinker [331] and I. J. Sands.[332] However, with the discontinuation of its administrtation, the seizures returned often at more frequent intervals and with greater intensity. As the remedy has no action in hysteria crises, Maillard [333] considers it an aid in the diagnosis.

Cheinisse,[334] on the other hand, warns that, though the drug acts well in the seizures, it also checks the normal activity of the nerve centers, entailing a physical and mental torpor. He cites Ducoste, who combines the drug with belladonna or caffein to ward off these by-effects, and mentions other authors who recorded negative therapeutic results and saw death in status epilepticus following sudden suspension of phenobarbital treatment. Weber [335] and Fremel, with Herschmann,[336] write on phenobarbital by-effects. The latter authors emphasize injuries to the cerebellar and vestibular system and comment on barbital psychosis.

Fischer [337] advances evidence that the capacity of the body to react with convulsions has a central and peripheral component, the latter having a relationship especially to the suprarenals. Diminution of their substance by extirpation is followed by reduction of convulsive reactions on stimulation. Enlargement of the suprarenal cortex as by castration or alcohol poisoning of rabbits, is followed by an increase of the spastic reaction.

Acting on this principal, Steinthal [338] operated on eleven epileptics, extirpating one of the suprarenals, but without convincing results.

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330. Roubinovitch, J., and Chavany, J. A.: Bull. Acad. de med. **85**:687 (June 7) 1921.

331. Grinker, J.: Phenobarbital in Epilepsy, J. A. M. A. **79**:788 (Sept. 2) 1922.

332. Sands, I. J.: Luminal Therapy in Epileptic Seizures, Arch. Neurol. & Psychiat. **5**:305 (March) 1921.

333. Maillard, G.: Bull. med. **35**:769, 1921.

334. Cheinisse, L.: Presse méd. **30**:42 (Jan. 14) 1922.

335. Weber, H.: Therap. Halbmonatsch. **35**:467 (Aug. 1) 1921.

336. Fremel, F., and Herschmann, H.: Med. Klin. **17**:716 (June 12) 1921.

337. Fischer, Heinrich: Deutsch. Ztschr. f. Nervenh. **71**:116, 1921.

338. Steinthal, C.: Zentralbl. f. Chir. **48**:878 (June 25) 1921.

Sándor [339] obtained in four cases a certain degree of benefit, which, on account of the short postoperative period, cannot be determined yet. Fischer [340] reports distinctive improvement in a case of long standing, and Kersten [341] in two epileptics.

Sultan [342] holds the prospects of a cure by this operataion as small, and still more skeptical is Specht,[343] who, from experimental research, denies justification of surarenalectomy in treatment of epilepsy.

PSYCHOLOGY

The biography of an infant prodigy, Chr. H. Heineken, born at Luebeck, 200 years ago, has been sketched by Brüning.[344] At the age of 14 months, he knew the whole Bible; at 2½ years, ancient history, geography, anatomy and 800 Latin words, learning weekly over 150 new vocables. He could read German and Latin and spoke German, low German, Latin and French perfectly. When 3 years old, he could add, subtract and multiply, and in his fourth year he learned 220 songs, eighty psalms, 1,500 verses and sentences of Latin writers. Shortly before his death, at the age of 4 years and 4 months, he studied astronomy.

Mosse,[345] when testing children in the kindergartens, encountered suggestibility at the early age of 3 years. Experiences at the dispensary demonstrated that the results of examinations of healthy children as to their suggestibility are not applicable to the sick child, since the suggestibility of the former decreases, that of the latter increases with age. The great suggestibility of children necessitates great caution in children's testimonies at law suits.

Among 70,555 school children of Vienna examined, Neurath [346] found lefthandedness existing in 3.85 per cent. His studies lead him to the conclusion that it is a congenital peculiarity, more frequent in the male than in the female, inherited directly, usually from the mother. Not only the left hand, but the total motor innervation area of the right hemisphere, as to skilfulness and spontaneous motility revealed a psychomotor preponderance and superiority over the right side of the body. The combination with disorders of speech is not infrequent. The left-handed persons represent not a type of degeneracy, but a variety of the human species, inherited from generation to generation. In pedagogic practice, the author postulates for the sinistrals a wide range of liberty.

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339. Sándor, S.: Zentralbl. f. Chir. **48**:881 (June 25) 1921.
340. Fischer, H.: Ann. Surg. **76**:176 (Aug.) 1922.
341. Kersten, H.: Med. Klin. **49**:1553 (Dec. 3) 1922.
342. Sultan, C.: Deutsch. med. Wchnschr. **48**:153 (Feb. 2) 1922.
343. Specht, O.: Beitr. z. klin. Chir. **125**:347, 1922.
344. Brüning, H.: Monatschr. f. Kinderh. **22**:230 (Nov.) 1921.
345. Mosse, K.: Ztschr. f. Kinderh. **27**:65, 1922.
346. Neurath, R.: Wein. klin. Wchnschr. **35**:895, 1922.

Sieben [347] analyzes sinistrality in five generations, and an editorial in *The Journal of the American Medical Association* dealing with this condition,[348] quotes C. Quinan as finding that among sinistrals stammering is three to six times more frequent than among dextrals.

SEXUALITY

That the conception of asexuality or presexuality in childhood is not tenable, becomes evident from the abundant material collected by Freud and his school. Friedjung [349] insists that the scope of sexual manifestations embrace not only instinct acts aiming directly at heterosexual union, but also pleasurable gratifications of Freud's so-called partial instincts, such as olfactory impulses and masochistic and sadistic tendencies. The earliest period of childhood is characterized by autoeroticism, originating in various erogenous zones, especially the lips, skin, anus and urethra. Cases of enuresis were traced to urethral eroticism. There is no escape for the child from experiencing sensations localized in the sexual organs, either by spontaneous erections which may occur very early or by the necessary washing processes. Masturbation has been observed in infants of 9 and 13 months. Among the manifestations of hetero-eroticism, the most frequent is seeking after and playing with the breast of the mother or some loved woman with excitement, observed in boys from the second to the fourth year of life; obviously passionate kissing of the naked arm or neck of adults, excitement displayed when seeing nude legs and other more aggressive acts. Naturally, sexual curiosity is early focused on the genitals, in particular of the opposite sex, and may lead to mutual exhibition and some more aggressive acts. The habit of adults of taking the child into their bed is very deleterious, and mothers are often alarmed at the behavior of their little boys. In a chapter on psychosexual conduct, Friedjung cites examples of the Oedipus complex, exhibition and curiosity. Morbid modesty at puberty had in some instances its source in fear to have onanism detected. It is significant that all conditions mentioned fall within the normal, and that the children offered later no deviations from the average.

There exists a false incontinence of the sphincters in children due to what Morichau and Beauchant [350] term paragenital auto-eroticism. They hold the urine or stool back on account of a pleasurable sensation then felt in the bladder, urethra or anus until this voluntary retention is overcome by the demands of Nature and the child soils his clothing.

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347. Sieben, W.: Deutsch. Ztschr. f. Nervenh. **73**:213, 1922.
348. Is Lefthandedness a Sign of Inferiority? Editorial, J. A. M. A. **76**:1010 (April 9) 1921.
349. Friedjung, J. K.: Ztschr. f. Kinderh. **31**:1, 1921.
350. Morichau-Beauchant, R.: Paris méd. **12**:83 (July 22) 1922.

Bed wetting existing in a youth of 18 since his seventh year of life had been an unconscious equivalent for a sexual act. This was brought out through the psychanalysis by Herbert,[351] and is in line with Freud's statement that in infancy excretory functions (urinary and alvine) are charged with a sexual tone.

Three interesting cases, in two boys and a girl, from 3½ to 5 years old, reported by Klaesi,[352] demonstrate how marked an imprint of sublimated spirituality the sexual life at this age may have and how, under certain conditions, it may assume the qualities of virtue, as modesty, devotion, self-sacrifice, chivalry and courage, as in the adult. The author explains that whether in such cases it comes to masturbation or intercourse or not depends merely on external circumstances, and such acts are not necessarily to be considered as diagnostic signs of psychopathy.

There is no doubt that, as Anton [353] states, pathologic instances of precocious puberty with signs of accelerated physical and mental development, are not rare. He quotes Meynert's experience that powerful impressions and experiences even in early age may become fixed, laying the foundations for perversities and sexual excitations that subjugate the nervous system to the instincts and render the emotional life morbid. Great caution is necessary in the selection of persons to educate children.

Obstinate onanism since earliest childhood in a girl of 10, observed by Ellison,[354] was cured through fear of a threatened operation on the genitals after admission to a hospital.

NEUROPSYCHOPATHIC DISORDERS

Two principal groups of psychic anomalies in early childhood are distinguished by Goett;[355] (1) defects in the intellectual sphere; oligophrenia, and (2) the psychopathic anomalies. The latter are within the field of emotions, volition and character; within the personality proper. The earliest sign may be dysthymia and cyclothymia. The active subform is characterized by exaggeration of the instinct of self-preservation, manifesting itself in egoism; the passive type, by the prevalence of phantastic dreaminess and brooding logic in comparison to the pleasures of the senses of the normal child. Not the adult but the healthy child of the same age must be the standard in determining what is normal or abnormal, and the boundary of normality should be widely drawn. Psychopathy must not be confounded with naughtiness. In the psychopathic child, the egoistic tendencies are unrestrained for

351. Herbert, S.: Psychoanalytic Rev. **9**:263, 1922.
352. Klaesi, J.: Ztschr. f. ges. Neurol. u. Psychiat. **74**:362, 1922.
353. Anton: Ethik. Paedagogik u. Hyg. d. Geschlechtslebens **1**:10, 1922.
354. Ellison, E. M.: New Orleans M. & S. J. **74**:160 (Sept.) 1921.
355. Goett, T.: Klin. Wchnschr. **1**:265, 1922.

lack of inhibition, rooted in its unfortunate "anlage." In the non-psychopathic, merely spoiled child, the egoism is unbridled because there exists no necessity for restraint.

Increasing attention is given to nervous disorders as the result of injurious environment, especially of the influence of persons around the child. A characteristic type is circumscribed as the "only child." Friedjung [356] observed among 264 "only children" not more than thirty-five that were healthy. Other types are the favorite children, such as the first born, or the late born child, the only boy among several sisters or vice versa. While these children suffer from exaggerated care, another group suffers from the opposite: such as the child that is not loved, that is among strangers, the foster child, the child that is less beautiful or talented, etc.

The importance of the development of the right kind of emotions by young persons and their correct guidance by the parent is emphasized by Williams.[357]

The polymorphous clinical picture in the nervous child is analyzed by Heubner [358] and exemplified by a number of instructive cases from his vast experience. The essential feature is the predisposition of the nervous system to react in an abnormal manner to the common stimulations of life. The nervous child often suffers from anorexia, pallor and headache. The pallor is not due to any hematologic anomaly but to a vasor disturbance; the headache can be traced mostly to a provocative cause; the anorexia may find its explanation in the lack of pleasure children, at least in the playing age, find in eating. Other manifestations are vomiting, abdominal pain, sleeplessness, enuresis, masturbation and nervous disturbances of the respiratory organs and the heart, and finally tics and similar motor disturbances. They all are discussed.

Many symptoms of so-called neuropathic diathesis may be due to anaphylactic reaction to food proteins. This is brought out by Shannon,[359] who, in eight cases of neuropathic diathesis in infants and children, proved protein sensitization by cutaneous tests made with a number of foods. All showed definite relief from nervous symptoms after exclusion of the offending food or following specific protein therapy.

Delinquency with its characteristic lack of self-control, of moral and ethic motives, loss of well-balanced, regulated conduct and of social

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356. Friedjung, J. K.: Wien. klin. Wchnschr. **34**:70 (Feb. 17) 1921; Deutsch. med. Wchnschr. **47**:949 (Aug. 18) 1921.

357. Williams, T. A.: Arch. Pediat. **39**:180 (March) 1922.

358. Heubner, O.: Klin. Wchnschr. **1**:980 (May 13) 1922.

359. Shannon, W. R.: Neuropathic Manifestations as a Result of Anaphylactic Reaction to Foods Contained in Their Dietary, Am. J. Dis. Child. **24**:89 (July) 1922.

adaptation, as again pointed out by Cimbal,[360] has its origin mostly in extraneous factors, such as lack of home and school education and demoralizing conceptions, though even an orderly home environment offers no absolute protection. Its manifestation at school age is mostly truancy; later theft and fraud, and at puberty a craving for stimulations by tobacco, exciting reading and shows and vagabondage. Mental deterioration may supervene, with impairment of memory, sometimes surpassing that of paralysis, when the distinction from juvenile insanity may be difficult. Predisposing factors are congenital inferiority and exhaustion of the nervous system.

To decide how far an innate mental anomaly and how far the child's environment is responsible for abnormal conduct reactions, Lurie[361] urges that the child be taken out of that environment and placed in a psychopathic institution especially fitted for such scientific observations.

Sundell[362] believes that sleeplessness in infants often has its cause in the nursery, especially in an irregular bed time and excitement while awake. These and other educational errors must be avoided. Hypnotics must be reserved for exceptional occasions, and then an adequate dose should be given for the first evening, a gradually lessened dose for the next two days and the fourth evening the hypnotic should be omitted. The various sedative and soporiferous remedies employed in childhood were the subject of an instructive paper by Rominger.[363]

De Haan[364] presents arguments that the main cause of enuresis is exceptionally profound sleep. All that is necessary to cure it is to render this sleep less profound at night by a nap during the day or by putting the child to bed a few hours earlier than usual, but without darkening the room till the usual time. In addition to this procedure, Zappert[365] recommends life in the country, psychotherapy reeducation of the bladder and reduction of sodium chlorid in the diet. Also potatoes, bread and vegetables should be lessened in quantity as they cause an increased output of potassium with a postponed elimination, taking place during the night, resulting in nycturia, polyuria and pollakiuria.

MIGRAINE

That migraine is not rare in children is the experience of Comby.[366] The hereditary predisposition is homologous and heterologous, since

360. Cimbal, W.: Deutsch. Ztschr. f. Nervenh. **68-69**:365, 1921.
361. Lurie, L. A.: Treatment of the Subnormal and Psychopathic Child, J. A. M. A. **71**:1386 (May 21) 1921.
362. Sundell, C. E.: Practitioner **109**:89 (July) 1922.
363. Rominger, E.: Klin. Wchnschr. **1**:1949 (Sept. 23) 1922
364. De Haan, P. B.: Nederl. Tijdschr. v. Geneesk. **2**:1666 (Oct. 1) 1921; abstr., J. A. M. A. **77**:2154 (Dec. 31) 1921.
365. Zappert, J.: Klin. Wchnschr. **1**:27 (Jan. 1) 1922.
366. Comby, J.: Arch. de méd. d. enf. **24**:29 (Jan.) 1921.

asthma, neuropathies, hysteria and arthritism may be traced in the ancestry; and the children themselves later may have neuroarthritic manifestations, such as asthma, diabetes, renal calculi or gout.

Curschmann [367] saw migraine as early as in the beginning of the second year of life. During school age, the affection is characterized by its association with abdominal symptoms of splanchnogenic origin. Navel colic, many cases of supposed appendicitis, of gastric or duodenal ulcers, etc., have been really attacks of migraine or its equivalent. There may even be fever present. The splanchnogenic origin of the abdominal pains becomes apparent from the paroxysmal rise of blood pressure. A number of instructive case histories illustrate the various clinical pictures. Anaphylactic shock and spasmophilia or parathyroid states seem sometimes to coexist. Specific antitetany remedies, such as calcium, prove of great benefit.

Certain gastralgias in the adult in connection with migraine, either accompanying the typical attacks or being of the vicarious or the larval, irregular type, have long been known. Only recently, Brams [368] has reported a number of his observations at the policlinic of Paul Cohnheim in Berlin; and in one of the seven cases with periodical abdominal crises, cited by Buchanan,[369] the patient had been operated on but no lesion was discovered.

That such epigastric pains are related to migraine and hypertension due to vascular spasms seems very probable from the experiences of Hadlich [370] that migraine and hypertension frequently occur in one and the same patient; or that hypertension exists in one and migraine in another member of the family.

It seems noteworthy that systematic ingestion of peptone before each meal and Bonche's method of weekly injections of horse serum or crotalin proved useful in certain cases of migraine as reported by Pagniez.[371] Loewenthal [372] saw beneficial results from treatment with radium emanations, and Sicard [373] and his co-workers noticed that following systematic injections of sodium carbonate, the migraine ceased in every instance.

PSYCHIC DEFECTS

Psychometric examinations made by Raeder [374] of twenty-two children of syphilitic parents showed psychic terracing pari passu, with the

367. Curschmann, H.: München. med. Wchnschr. **69**:1747 (Dec. 22) 1922.
368. Brams, W. A.: Abdominal Migraine, J. A. M. A. **78**:26 (Jan. 7) 1922.
369. Buchanan, J. A.: J. Nerv. & Ment. Dis. **54**:406 (Nov.) 1921.
370. Hadlich: Deutsch. Ztschr. f. Nervenh. **75**:125, 1922.
371. Pagniez, P.: Presse méd. **29**:45 (Jan. 15) 1921.
372. Loewenthal, S.: München. med. Wchnschr. **67**:971 (June 30) 1922.
373. Sicard, J. A., Parafand, Forestier: Bull. et mém. Soc. méd. d. hôp. de Paris **45**:1204 (July 22) 1921; abstr., J. A. M. A. **77**:1052 (Sept. 24) 1921.
374. Raeder, O. J.: Feeblemindedness in Hereditary Neurosyphilis, Am. J. Dis. Child. **21**:240 (March) 1921.

grades of physical defects. The virulence of the congenital syphilis toxin in a given family of children, as evidenced by psychometric analysis and biochemical tests, varied inversely with the order of birth.

There is evidence accumulating that an acquired iodiocy of parathyroid origin is not uncommon. Comby [375] states that fortunately these cases are amenable to parathyroid treatment.

Gordon [376] found lefthandedness in 18.2 per cent. of children in schools for the mentally defective. It was often associated with defects of speech. The explanation given is that something had occurred interfering with proper functioning of the dominant hemisphere, the same cause affecting the higher intellectual centers, assumed to be localized in the same hemisphere. Mirror writing was more often encountered than in pupils in public schools.

True mental disease is extremely rare in childhood. Excluding postinfectious mental disturbances, juvenile paresis, epileptiform phenomena, psychoneurosis, constitutional psychopathy and mental defectiveness, only eighteen instances of true psychosis in 5,000 hospital admissions were observed by Strecker [377] in children under 15 years of age. Four were of indeterminable character; in ten the diagnosis was manic-depressive psychosis, in four dementia praecox. The symptomatology is essentially more simple in children than in the adult, presenting elementary emotional reactions and trivial delusions. Alexander is quoted as stating that between 10 and 30 per cent. of psychoses in children are due to acute infectious diseases, especially exanthemas, and recently also epidemic encephalitis.

Schnabel's [378] review of 231 cases of psychic disturbances at the ages of from 2 to 16 years, observed at the psychiatric clinic at Zürich, demonstrates the good prognosis of hysteria. Then follow the constitutional psychoses, the psychopathic conditions and moral defectiveness. On the other hand, the prognosis in schizophrenia and epilepsy is unfavorable, and in hebephrenia associated with weakmindedness very grave.

Zappert [379] reports seven cases of infantile dementia, an affection to which Heller first called attention in 1908. The symptoms appear in the third or fourth year of life, with disturbances of speech, which later becomes much aggravated until spontaneous speech and understanding of the heard word ceases. There exist restlessness, hallucinations, states of fear and dementia, that within a few months ends in complete idiocy, the condition finally becoming stationary.

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375. Comby, J.: Arch. de méd. d. enf. **24**:303 (May) 1921.
376. Gordon, H.: Brain **43**:313 (Jan.) 1921.
377. Strecker, E. A.: New York M. J. **114**:209 (Aug. 17) 1921.
378. Schnabel, J.: Ztschr. f. d. ges. Neurol. u. Psychiat. **68**:241, 1921.
379. Zappert: Monatschr. f. Kinderh. **22**:389, 1921.

The studies of the therapeutic effects of tuberculin and typhoid vaccine and especially of artificially inoculated malaria tertiana in progressive paralysis (dementia paralytica), as they have been carried out at the psychiatric clinic at Vienna for the last few years, are of considerable interest. Following the communication by Wagner-Jauregg [380] on this subject, Gerstmann [381] in his last report, gives a brief review of the results in thirty-nine patients treated from 1917 to May, 1920, with the plasmodium. Since September, 1919, 200 patients, most of them having dementia paralytica, and recently also patients having locomotor ataxia or tertiary and congenital syphilis were inoculated with tertiane plasmodium. After from nine to twelve malarial attacks, quinin-neoarsphenamin treatment is instituted. Among 116 cases studied thoroughly, thirty-eight cases, far advanced and of long standing, remained unimproved. Forty-two instances manifested complete remissions without any residual signs of psychic defect and thirty-six patients incomplete remissions with some residual symptoms. Extremely interesting observations were made, such as sudden alteration of the psychotic picture following the malaria attacks, and acute reactive intensification of the paralytic symptom complex until the remission took place after the antimalarial treatment. Encouraging results are reported also by Weygandt.[382]

MONGOLIAN IDIOCY

That mongolian imbeciles are often the last children of large families and physically exhausted mothers is reiterated by Comby.[383] He recalls the fact that they are more frequent among nations with a high than those with a low birth rate, such as the French. The first two claims are borne out also by the experiences of von Hofe.[384]

On the other hand, H. Thursfield [385] in his study of forty-two mongolians, finds no corroboration of the current statement that the mongoloid is apt to be the last child of a long family. He notes, however, that in six instances previous to the birth of the idiot the mothers had failed to bear children for a long period.

Stolzner's view of an existing causal connection between hypothyroidism of the mothers during pregnancy and mongolism is not borne out by Dollinger [386] who, on the contrary, in nineteen mothers, could establish anamnestically a slight hyperthyroidism. The author notes the

380. Wagner-Jauregg: Wien. klin. Wchnschr. **34**:171 (April 14) 1921
381. Gerstmann, J.: Ztschr. f. ges. Neurol. u. Psychiat. **74**:242, 1922.
382. Weygandt: Deutsch. med. Wchnschr., 1921, p. 1013.
383. Comby, J.: Medicine **2**:835 (Aug.) 1921.
384. Von Hofe, F. H.: Arch. Pediat. **39**:737 (Nov.) 1922.
385. Thursfield, H.: Brit. J. Dis. Child. **18**:18 (Jan.-March) 1921.
386. Dollinger, A.: Ztschr. f. Kinderh. **27**:332 (Jan.) 1921.

increasing frequency of this anomaly and calls to mind that the theory of exhaustion by advanced age should be extended to the fathers.

An argument against a causative relationship between myxedema and mongolism is the fact brought out by Nadolny and Weinberg [387] that the blood picture in the latter shows no deviation from the normal and no similarity with the findings in myxedema; and that the basal metabolism, studied by Fleming [388] in six mongoloids, was normal and not affected by thyroid treatment.

Peculiar changes in the anterior portion of the fossa pituitaria consisting in an excavation under the anterior clinoid processes and optic groove have been described by Timme.[389] This condition in conjunction with certain clinical signs suggest to him a disturbance of the anterior part of the pituitary body as the cause of many of the symptoms of mongolism.

Mongolian idiocy in one of twins, a rare occurrence, has been reported by McLean [390] and Clay.[391] Clay invites investigation on the part of the obstetrician, as the determination of the monozygotic or dizygotic origin of such twins may aid in determining whether the cause of mongolian imbecility, occurring in one of twins, has a maternal or paternal etiology. Halbertsma [392] in connection with five new cases and nine from the literature on mongolianism in one of twins, advances the hypothesis that the underlying factor is malformation of the median part of the skull, a "vitium primae formationis" due to an endogenic element. The fact that among double-ovum twins the one is entirely normal seemed to point to an endogenous factor and the usually advanced age of the mother to the anomaly having its origin in the ovum. The occurrence of mongolian imbecility in the Mongolian race (Chinese) has been confirmed by a case report of Demuth [393] and Tumpeer.[394]

The anatomic pathology in a baby of 21 months described by Cozzolino [395] was the following: underweight brain, asymmetry of hemispheres, reduction of the thickness of cortex and of the number and extent of convolutions; marked underdevelopment of the lower frontal convolutions and an extension of the parieto-occipital fissure.

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387. Nadolny, G., and Weinberg, M.: Ztschr. f. Kinderh. **29**:68 (April) 1921.
388. Fleming, G. B.: Quart. J. Med. **16**:11 (Oct.) 1922.
389. Timme, W.: Mongolian Idiot; a Preliminary Note on Sella Turcica Findings, Arch. Neurol. & Psychiat. **5**:568 (May) 1921.
390. McLean, S.: Mongolian Idiocy in One of Twins, J. A. M. A. **78**:13 (Jan.) 1922.
391. Clay, H. T.: Arch. Pediat. **39**:726 (Nov.) 1922.
392. Halbertsma, T.: Nederlandsch Tijdschr. v. Geneesk. **2**:22, 1922; Zentralbl. f. ges. Kinderh. **13**:465, 1922 (abstr.).
393. Demuth, F.: Ztschr. f. Kinderh. **33**:110 (July) 1922.
394. Tumpeer, T. H.: Mongolian Idiocy, J. A. M. A. **79**:14 (July 1) 1922.
395. Cozzolino: Pediatria **29**:49 (Jan. 15) 1921.

CEREBROSPINAL FLUID

The fact that Dandy produced unilateral internal hydrocephalus by obstructing one foramen of Monroe and that extirpation of the choroid plexus in such an obstructed lateral ventricle prevented the development of internal hydrocephalus is declared by Weed [396] to be the strongest single piece of evidence that the origin of the cerebrospinal fluid is in the plexuses. However, there exists anatomic evidence that the perivascular spaces, the ependymal lining of the ventricles and the central canal of the spinal cord contribute a minimum addition to the fluid.

The constant amount of calcium in the liquor under physiologic and pathologic conditions, as found by Leicher [397] seem to indicate that the fluid is a secretion.

Xanthochromia has been reported in about 310 cases in the literature in connection with hemorrhages in the central nervous system and its membranes, hemorrhagic inflammation and pressure on the cord, whenever erythrocytes had found their way into the spinal fluid. The color, as demonstrated by Leschke [398] is due to bilirubin, formed from hemoglobin.

The presence of hemolysins in lumbar puncture fluid has been found by Schleissner [399] to be a reliable index for the presence of purulent meningitis and for the trend of recovery or aggravation. He recommends the greater use of Weil-Kafka's method by the pediatricians.

For the not infrequent cases in which only small quantities of cerebrospinal fluid are available, special micromethods have been worked out for cell count and various reactions. Detailed directions for various micromethods with a material varying from 1 to 15 drops are given by Plaut.[400]

A simple methylene-blue method for the qualitative and quantitative determination of the reducing substance in the cerebrospinal fluid is described by Michio Kasahara, and Shunjiro Hattori.[401] It is rapid, convenient and sufficiently accurate for practical use and is recommended also by Strong [402] for the less elaborately equipped laboratory.

In order to facilitate differentiation in the counting of lymphocytes from erythrocytes admixed in the spinal fluid, Jacobsthal [403] recom-

396. Weed, L. H.: Psysiol. Rev. **2**:171 (April) 1922.
397. Leicher, H.: Deutsch. Arch. f. Klin. Med. **141**:196, 1922.
398. Leschke, E.: Deutsch. med. Wchnschr. **47**:376 (April 7) 1921.
399. Schleissner, F.: Med. Klin. **17**:1206 (Oct. 2) 1921.
400. Plaut, F.: Ztschr. f. ges. Neurol. u. Psychiat. **65**:373, 1921.
401. Kasahara, Michio, and Hattori, Shunjiro: Rapid Clinical Method for Determination of Reducing Substance in Cerebrospinal Fluid; Methylene Blue Method, Am. J. Dis. Child. **22**:218 (Aug.) 1921.
402. Strong, R. A.: Arch. Pediat. **39**:431 (July) 1922.
403. Jacobsthal, E.: Deutsch. med. Wchnschr. **48**:867 (June 30) 1922.

mends an increase in the concentration of the methylene-violet and the acetic acid according to his formula, which is given. The interferometer method of Loewe for determining the concentration of the cerebrospinal fluid as a diagnostic means in affections of the central nervous system is approved by Wüllenweber.[404]

The value of the routine use of the colloidal gold reaction in acute poliomyelitis becomes evident from the study of Regan and Cheney.[405] In every case during the acute stage there was a reaction in the "syphilitic zone," with typical rising and falling curves for the first and second weeks, thereafter gradually subsiding. The reaction may be of some use in the differential diagnosis of epidemic encephalitis. In conjunction with the clinical findings, the test may be the most valuable laboratory method we possess for the recognition of poliomyelitis. The authors believe that when the curve becomes normal, the acute period is over, and the more energetic treatment of the subacute stage may begin.

Of practical importance are the findings of Baar [406] that the globulin content may be increased in uremic-eclamptic attacks, and equal that in tuberculous meningitis, especially in repeated spasmophilic convulsions and in a number of organic affections of the central nervous system. A negative Pandy's reaction, excluding an increase of globulin, argues against tuberculosis in the differential diagnosis from clinically similar functional disorders. For the differential diagnosis between tuberculosis and other organic affections of the central nervous system with a similar symptom complex, the positive reaction is of little value, except that a continuous increase of its intensity would indicate tuberculosis. Aschenheim,[407] referring to the paper on Pandy's reaction by Widmaier,[408] cautions against an overvaluation of a positive Pandy's reaction for the diagnosis of tuberculous meningitis.

On account of the frequency of cerebrospinal involvement in prenatal syphilis, Kingery [409] stresses the importance of routine lumbar punctures.

The studies of comparative results of colloidal mastic and colloidal gold tests carried out by Keidel and Moore [410] with spinal fluid of 311 mostly syphilitic patients, warrant a wider use of the former than is now made, since it seems to detect anomalies more frequently than does the

404. Wüllenweber, G.: München. med. Wchnschr. **69**:927 (June 23) 1922.

405. Regan, J. C., and Cheney, G. W. H.: Value of Use of Colloidal Gold Reaction in Acute Epidemic Poliomyelitis, Am. J. Dis. Child. **23**:107 (Feb.) 1922.

406. Baar, H.: Wien. klin. Wchnschr. **34**:614, 1921.

407. Aschenheim, E.: München. med. Wchnschr. **68**:1015 (Aug. 12) 1921.

408. Widmaier, E.: München. med. Wchnschr. **68**:772 (June 24) 1921.

409. Kingery, L. B.: Study of Spinal Fluid in Congenital Syphilis, J. A. M. A. **76**:12 (Jan. 1) 1921.

410. Keidel, A., and Moore, J. E.: Comparative Result of Colloidal Mastic and Colloidal Gold Tests, Arch. Neurol. & Psychiat. **6**:163 (Aug.) 1921.

gold test. The liquor findings in congenital syphilis were studied by Tezner [411] with sixty-two punctures in forty-three children. In 41 per cent. the findings were positive. The Wassermann reaction was positive in 32 per cent. Pandy's test showed parallelism with Nonne's; other reactions lacked parallelism. The findings are evanescent and do not indicate a permanent injury to the central nervous system.

Not only an excessive quantity of cerebrospinal fluid, with its increased intracranial pressure, but also hypotension associated with too little cerebrospinal fluid is liable to cause symptoms, as headache, vertigo, convulsions and torpor. Leriche [412] describes a few illustrative cases demonstrating this, and the beneficial effect of return to normal cerebrospinal pressure by cutaneous saline infusions. Similar symptoms occasionally followed lumbar puncture. Convulsions in children should, therefore, be studied with these considerations in view.

That lumbar puncture may involve serious dangers is emphasized by Guillain and Laroche,[413] especially in Pott's disease. They quote Sicard, who avoids puncture in bulbar affections.

Caronia and Auricchio [414] found the specific bacillus in typhoid and paratyphoid so regularly in the cerebrospinal fluid that they see in the examination a valuable diagnostic method in suspected typhoid.

REFLEXES

The statement generally made that changes in the classic deep reflexes have little diagnostic value in early infancy is borne out in the study by Burr [415] of sixty-nine normal infants. Among the reflexes, the plantar was the most variable, and the Achilles tendon reflex the most frequently lacking. Feldman [416] failed to obtain a plantar response in only 15 per cent. of 500 children from birth up to 7 years of age. While all observers, except Finizio, find the prevailing response of Babinski type in the very young, he found the proportions between plantar flexion and dorsiflexion to be 4 : 1. In premature infants, the response is nearly always of the Babinski type for from five to six weeks of postnatal life, because of the absence of myelinization of the pyramidal tracts. The definite disappearance of Babinski's type depends in his experience by no means on the age at which the child begins to walk.

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411. Tezner, O.: Monatschr. f. Kinderh. **22**:4, 1921.
412. Leriche, R.: Lyon. chir. **19**:57 (Jan.-Feb.) 1922.
413. Guillain, G., and Laroche, G.: Bull. et mém. Soc. méd. d. hôp. de Paris **45**:794 (May 27) 1921.
414. Caronia, G., and Auricchio, L.: Pediatria **30**:337 (April 15) 1922.
415. Burr, C. W.: Reflexes in Early Infancy, Am. J. Dis. Child. **21**:529 (June) 1921.
416. Feldman, W. M.: Brit. J. Child. Dis. **18**:24 (Jan.-March) 1921.

The contradictory reports on the oculocardiac reflex (Dagnini-Aschner) induced Sante Naccarati [417] to study it in normal and pathologic subjects. Some of his conclusions are that the reflex cannot constitute a positive sign for differential diagnosis, and that it may serve only as an indicator of probability. Large deviations from the reflex index may under certain conditions indicate abnormal states. In tabes, for instance, the reflex index is 0 or very small.

Resek [418] succeeded in provoking for the first time a conditional reflex (Babinski's). All laws of a conditional reflex pertaining to alterations of the stimulation inhibition and extinction were proved to exist.

Children of early school age, when asked to open the mouth wide for inspection, frequently exhibit peculiar associated movements to which Goett [419] calls attention. They consist in a stiffening of the body, marked abduction (spreading) of the fingers, with the arms stretched downward and the eyes opened wide. The movements are related to expressive movements and are induced by the subconscious idea of "opening" (mouth, eyes, fingers).

MISCELLANEOUS

Venipuncture of the superior longitudinal sinus appears to be the best method for obtaining blood from the new-born or for intravenous medication in infants. Gordon,[420] in 231 punctures, experienced no untoward results. At the Rostock children's clinic, it has been performed 3,000 times, as Krasemann [421] reports. For facilitating the procedure, Ratner [422] devised a new apparatus.

Wentzler [423] constructed a tonometer which, applied over the anterior fontanel, serves for the measurement of the intracranial pressure.

In a statistical survey of 100 cases of skull fractures in children, Moorhead and Weller [424] established a mortality of 17 per cent., associated injuries excluded. The mortality with involvement of the base was four times that of vault injuries alone. More than three fourths of the fatalities occurred in the first two days. In comparison with adults, children have a 25 per cent. better chance for life with the same grade of skull injury.

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417. Naccarati, Sante: Oculocardiac Reflex (Dagnini-Aschner phenomenon) Its Use in Medicine and Psychology, Arch. Neurol. & Psychiat. **5**:40 (Jan.) 1921.
418. Resek, F.: Ztschr. f. Kinderh. **29**:85 (April) 1921.
419. Goett, T.: Ztschr. f. ges. Neurol. u. Psychiat. **66**:93, 1921.
420. Gordon, J. W.: Venipuncture of Superior Longitudinal Sinus in New-Born; Results in 231 Cases, J. A. M. A. **77**:1721 (Nov. 26) 1921.
421. Krasemann, E.: Med. Klin. **17**:809 (July 3) 1921.
422. Ratner, A. B.: New Apparatus and Method for Puncturing the Superior Longitudinal Sinus, Am. J. Dis. Child. **21**:199 (Feb.) 1921.
423. Wentzler, E.: Arch. f. Kinderh. **70**:241 (Jan.) 1922.
424. Moorhead, J. J., and Weller, W.: Ann. Surg. **74**:72 (July) 1921.

The peculiarities of the chemical composition of the infantile brain were studied by Schiff and Stransky [425] in twelve cases. It was found that the water content diminishes with age, and that the dry substance of the infantile brain contains more albuminous substances and less lipoids than that of the adult. The cholesterin in infants is more than double that in the adult, and the unsaturated phosphatids are less than in the adult. On account of the large amount of proteins that are capable of inhibition and the scarcity of lipoids that inhibit it, the infantile brain is in a condition very favorable for inhibition, to which possibly the predisposition to convulsions at this age may have a relationship.

Cephalic bruit in infants and children was the subject of study by Still.[426] No connection with any particular disease could be established. The phenomenon is possibly due to the temporary tortuosity of the carotids at the base of the skull, and the occasional persistence of audibility beyond the usual age may be related to an unusual thinness of the bony wall of the carotid canal.

Paralysis in a number of children due to the bite of wood-tick (*Dermacentor venustus*) has been reported by McCornack.[427] The condition may issue fatally from respiratory failure if the large, engorged wood-tick, found mostly in the scalp, is not removed entirely. Otherwise, recovery is rapid.

An outbreak of botulism affecting eight persons and ending fatally in six has been reported by Whiteman and Wilkinson.[428] Two children were among the victims. They showed decided weakness of the arms, legs and neck, slight ptosis and dysphagia. Recovery was slow. Colver,[429] who saw five cases of botulism, two terminating fatally, recalls that the affection is a toxic encephalitis localized in the various nuclei of the pons and in the medulla, probably also in the thalamus opticus, and that clinically it may be confounded with epidemic encephalitis, methyl alcohol poisoning, poliomyelitis and meningitis.

An elaborate monograph on tachymeningitis hæmorrhagica interna in childhood with numerous illustrations and twenty-two case histories has been published by Rosenberg.[430]

Total hemihypertrophy of the body with mental defect, one of the rarest developmental anomalies, has been reported by Gesell [431] in a

425. Schiff, E., and Stransky, E.: Jahrb. f. Kinderh. **96**:245, 1921.

426. Still, G. F.: Brit. J. Child. Dis. **18**:173 (Oct.-Dec.) 1921.

427. McCornack, P. D.: Paralysis in Children Due to Bite of Wood-Ticks, J. A. M. A. **77**:260 (July 23) 1921.

428. Whiteman, R. T., and Wilkinson, E. A.: Outbreak of Botulism, J. A. M. A. **78**:1278 (April 29) 1922.

429. Colver, B. N.: J. Michigan M. Soc. **20**:385 (Oct.) 1921.

430. Rosenberg, O.: Ergebn. d. inn. Med. u. Kinderh. **20**:549-638, 1921.

431. Gesell, A.: Hemihypertrophy and Mental Defect, Arch. Neurol. & Psych. **6**:400 (Oct.) 1921.

boy. A comprehensive tabulation shows a total of forty cases recorded in the medical literature, in five of which the patients were mentally defective. In Noronha's [432] case, the right side of the face was the larger and the trunk and limbs were larger on the left.

Posey [433] reports three cases of myasthenia gravis and Schäffer, with Brieger,[434] studied muscle tests in this affection.

Recklinghausen's disease has been reported by Parker [435] and Levin.[436]

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432. Noronha, A. J.: Indian M. Gaz., Calcutta **56**:10 (Jan.) 1921.
433. Posey, W. C.: Myasthenia Gravis, J. A. M. A. **77**:1077 (Sept. 24) 1921.
434. Schäffer, H., and Brieger, H.: Deutsch. Arch. f. klin. Med. **138**:28, 1921.
435. Parker, H. L.: J. Nerv. Ment. Dis. **56**:441 (Nov.) 1922.
436. Levin, O. L.: Recklinghausen's Disease: Its Relation to the Endocrine System, Arch. Dermat. & Syph. **4**:303 (Sept.) 1921; Arch. Neurol. & Psych. **8**: 690, 1922 (abstr.).

ACUTE HEMORRHAGIC PANCREATITIS RESULTING FROM ROUNDWORMS

REPORT OF CASE *

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Acute hemorrhagic pancreatitis in childhood is a rare disease. The case which is here reported is the only one that has been known to occur in the medical and surgical practice of the Hospital for Sick Children in a period of forty years. The reason for this rarity is evident when one considers that the conditions to which pancreatitis is usually attributed, such as gallstones and cholecystitis, are diseases of adult life.

The manner in which these conditions give rise to pancreatitis, or more specifically, cause trypsinogen of the pancreas to be converted into trypsin before it reaches the intestinal canal has been the subject of much investigation. It seems probable that the release of trypsin in the pancreas may be brought about by the introduction of either bacterial infection, through the lymphatics, blood stream or the pancreatic ducts, or of bile or duodenal juices, as the result of abnormal conditions at the ampulla of Vater. It has been demonstrated by Archibald and others that the injection of normal bile or intestinal juices into the pancreatic ducts will set up violent hemorrhagic pancreatitis, and cases have been recorded in which abnormalities at the ampulla, such as impacted gallstones, readily explain how it would be possible for a backwash of bile into the pancreas to occur. Two cases of severe pancreatitis have been described in which roundworms entered the ampulla and worked their way into the pancreatic duct. In one of the cases, described by Sir Hugh M. Rigby,[1] the condition was found at necropsy, and in the other, the patient was operated on by Novis,[2] and the roundworm removed. The successful result in the latter case appeals to us as a fitting reward for a masterly piece of surgery.

The case we have to record is another instance of pancreatitis produced, in all probability, by invasion of the ampulla by a roundworm.

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* Received for publication, Nov. 7, 1923.
* Read at the first annual meeting of the Canadian Society for the Study of Diseases of Children, Montreal, June, 1923.

1. Rigby, H. M.: Brit. J. Surg. **10**:419 (Jan.) 1923.
2. Novis: Brit. J. Surg. **10**:421 (Jan.) 1923.

The case is unique in that it occurred in a younger patient than any hitherto mentioned in the literature.

REPORT OF CASE

History.—A boy, aged 2½ years, thirty-six hours previously, at 6 a. m., began to cry with severe pain in the abdomen, doubling over and turning pale. He vomited in a few minutes and continued to vomit frequently until 3 p. m., when the pain, which had been continuous up to this time, ceased. The child was seen by Dr. Hewitt of Dunnville at 7 a. m., an hour after the onset of the symptoms, and a diagnosis of acute indigestion was made. He was seen again in the afternoon, and was given two simple enemas and later a milk and molasses enema, which were not effectual. A diagnosis of acute intestinal obstruction was made, and the patient was sent to Toronto. There was no history of intestinal worms and no report of previous illness except a mild attack of influenza about one week before.

Physical Examination.—The child was crying. The upper right side of the abdomen and the epigastrium were moderately rigid and apparently very tender. Crying was continuous, so that a satisfactory abdominal examination was impossible. There was no sign of shock. The child sat up in bed and cried lustily and fought against examination. Rectal examination was negative except that considerable gas of a fecal odor was passed. Under anesthesia, the abdomen was seen to be moderately distended. Running transversely across the abdomen, just below the level of the pylorus, a distinct tumor could be felt, firmly fixed in the posterior abdominal wall, about the thickness of one's thumb and about 3½ inches (8.7 cm.) long. No abnormality was noted elsewhere. The white blood cells numbered 15,000; polymorphonuclears, 68 per cent. Diagnosis: Partial intestinal obstruction with peritonitis from some unknown cause was diagnosed. The tumor was suggestive of intussusception, but it was too thin and too fixed, and the idea was abandoned before operation.

Operation.—At midnight, Feb. 15, 1923, forty hours after the onset of symptoms, the abdomen was opened by a right rectus incision. A small amount of blood stained fluid escaped, and considerable more was found among the coils of intestine. The transverse colon and great omentum were decidedly inflamed, and on the latter were observed numerous spots of fat necrosis about the size of a grain of wheat. Routine abdominal examination showed the appendix, cecum, small intestine and sigmoid colon normal. The gallbladder was distended, but showed no evidence of disease; it was easily emptied and contained no stones. The bile ducts seemed normal. The liver and stomach were normal. The tumor was then explored, and was found to be the pancreas covered with a mass of acutely inflamed swollen omentum. The mass was hemorrhagic and studded with dozens of patches of fat necrosis about the size of grains of wheat. All the peritoneum in the region of the pancreas and transverse colon, and also the great omentum and the neighboring small intestines were reddened and slightly sticky.

The blood stained fluid was mopped out of the peritoneal cavity. Drainage of the gallbladder was decided against. The abdomen was closed.

Course.—Following operation, the child slept quietly. There was no further pain or vomiting. During the day, the bowels were moved with an enema. At 3 p. m., i. e., fifteen hours after the operation, the patient was noticed to be gagging, and the nurse extracted a full sized roundworm from his mouth. Recovery was uneventful, and the child left the hospital at the end of ten days perfectly well.

COMMENT

We are of the opinion that the symptoms were produced by the roundworm, which had either introduced its head into the ampulla of

Vater, thus allowing a back flow of bile or intestinal juices into the pancreas, or had created such an irritation in the duodenum that the ampulla became closed off by the resulting inflammatory reaction. The symptoms of abdominal pain, shock, vomiting and obstruction resulted from the pancreatitis and peritonitis so produced. We believe that the disappearance of the pain and vomiting at 3 p. m. on the day of the onset of symptoms indicated that the cause of the pancreatitis ceased to be active at that time, and at this moment the roundworm ceased to obstruct the ampulla.

We were surprised to learn that further treatment for roundworms failed to demonstrate any more worms.

DR. LUTHER EMMETT HOLT

Dr. L. Emmett Holt, since its foundation a member of the editorial board of the AMERICAN JOURNAL OF THE DISEASES OF CHILDREN, died, January 14, in Peking, China, where he had gone to deliver a course of lectures at the Union Medical College. An obituary notice will appear in the March issue.